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American Journal of Diseases of Children

Vol. 10 DECEMBER, 1915 No. 6

DUODENAL ULCER IN INFANCY AN INFECTIOUS DISEASE *

LINTON GERDINE, M.D., AND HENRY F. HELMHOLZ, M.D.
CHICAGO

Since the publication[1] of the first large series of cases of duodenal ulcers in infants in 1909, numerous reports from various clinics and hospitals have appeared which have substantiated the frequent occurrence of duodenal ulcers in infancy. In 1913, Holt[2] collected ninety-one cases from the literature and added four more from his clinic. Since that time there have been numerous other publications on the subject which have increased the number of cases reported in the literature, but have not materially enriched our knowledge of the subject. Practically all the publications pass over the etiology of the ulcers with merely a reference to the usual explanations of the incidence of the ulcers as given in the textbooks of pathology (Holt,[2] Veeder[3]).

The etiology of gastric and duodenal ulcer is still a much debated question, so that any information as to its mode of incidence is of value. A very important point in the consideration of the etiology of the ulcers that has hitherto been overlooked is the fact that they have appeared in epidemic form, large series of cases occurring in a relatively short period of time. For example (1), in the first series of cases reported by one of us[1] in 1909, twelve cases were observed during the last four months of 1908, at the Berliner Kinder Asyl. In the first eight months of 1908, and in the entire year 1909, there were no further cases of ulcers observed at the Asyl. (2) Of his four cases Holt[2] says "Curiously three of these cases were observed within a period of three months." The fourth case was the only other one in a series of 1,800 necropsies. It can be added at this point that unless especially looked for, these ulcers are easily overlooked; but even if a

* Submitted for publication Oct. 15, 1915.

* From the Otho S. A. Sprague Institute Laboratory of The Children's Memorial Hospital.

1. Helmholz, H. F.: Duodenal Geschwüre und Pedatrophie, Deutsch. med. Wchnschr., 1909, i, 534.

2. Holt, L. E.: Duodenal Ulcer in Infancy, Am. Jour. Dis. Child., 1913, vi, 381.

3. Veeder, B. S.: Duodenal Ulcer in Infancy, Am. Jour. Med. Sc., 1914, cxlviii, 709.

few were overlooked in this series, the grouping of cases still remains very evident. (3) During seven months from September to April, we observed at this hospital eleven cases of duodenal ulcer. These eleven cases represent the only cases of duodenal ulcer that have been observed at our hospital.

This grouping of cases suggests very strongly that we are dealing with an infection and that we must seek for an explanation that will account for the epidemic-like appearance of ulcer cases. In his recent work, Rosenow[4] has pointed out new methods of studying ulcer cases which we have applied not only to our present series of cases, but also to great advantage to those cases described by one of us six years ago.

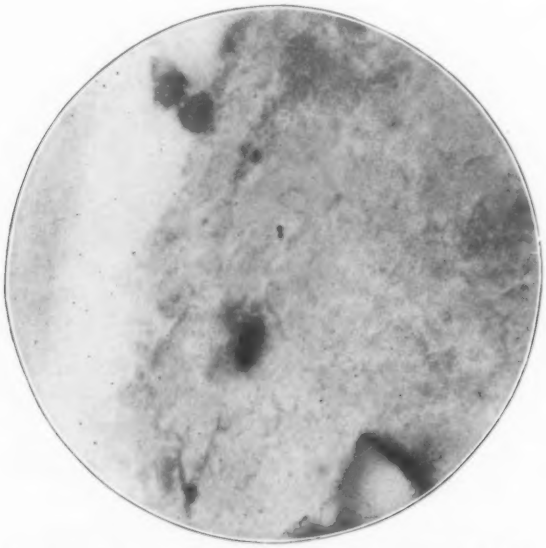

Fig. 1.—Photomicrograph of section from Case 1.

The following is a brief statement of the eleven ulcer cases, including clinical history, necropsy, and microscopic findings, as well as the examination for bacteria. The cases in which cultures were made from the ulcers at necropsy are also indicated. All the cases were observed between Sept. 24, 1914, and April 14, 1915.

REPORT OF CASES

CASE 1.—W. H. B., a male infant 3 months of age, was admitted to the hospital Oct. 21, 1914. The birth was normal. He was breast fed three weeks and then bottle fed at irregular intervals. He was never very well after he was 3 weeks old. The present illness was of about three weeks' duration;

4. Rosenow, E. C.: Bacteriology of Ulcer of the Stomach and Duodenum in Man, Jour. Infect. Dis., 1915, xvii, 219.

diarrhea and vomiting. One week before admission he was given boiled skimmed milk and water, 1½ ounces of each, every three hours. For a few days he improved. On the day of admission he passed blood in large amounts from the bowels. On admission the patient was in a state of collapse. He continued to pass a large quantity of dark blood from the bowels and died in less than twelve hours, Oct. 22, 1914.

Necropsy.—The body is that of a markedly emaciated male baby. The stomach is slightly thickened; no ulceration. On opening the peritoneal cavity there is seen a circumscribed peritonitis about the duodenum. The duodenum just beyond the pyloric ring shows a submucous hemorrhage measuring about ½ cm. in diameter. Just beyond the hemorrhage is a region of induration involving the duodenum on posterior and outer portion of first curvature and extending about three-quarters around the duodenum. In the center of the region of induration is seen a clear cut perforation of the wall measuring ¾ by ¼ cm. and running parallel to the duodenum. The ulcer measures 2½ by 1½ cm. The base of the ulcer is smooth, white, and is covered with black clots of blood. At its lower end is a lateral extension measuring ½ by 2/10 cm. There is dark tarry blood throughout the entire intestinal canal.

Anatomic Diagnosis.—Perforated duodenal ulcer, melena, encapsulated peritonitis.

Microscopic Examination.—Section stained with hematoxylin and eosin: The ulcer is rather shallow for about two-thirds of its extent and then dips down involving the circular muscular layer. In this area a large vessel is completely surrounded by necrotic tissue; its wall, however, is still intact. In the shallow portion of the ulcer there is a very wide zone of necrosis in which all structure is lost. The ulcer is characterized by more infiltration than is usually seen in the deeper layers of the duodenum.

Section stained for bacteria by the Gram-Weigert method: The section shows very beautifully several pairs of diplococci and numerous other cocci in the tissue below the surface of the ulcer in its deepest portion. Figure 1 is a photomicrograph taken from this section.

The following three cases are of particular interest because of the association with tuberculosis. In our previous series there was one case of tuberculosis that had numerous acute duodenal ulcers. We wish to separate these three cases from the remainder, however, because two were in older children and because the anatomic specimens were lost and there is a remote possibility that these ulcers were of tuberculous origin.

CASE 2.—S. F., a female child 10 years of age was admitted to the hospital Dec. 12, 1914. She had been a normal, healthy child. Had scarlet fever, chicken-pox and measles about 3 years before. The present illness was of about two weeks' duration. It began with sharp basal headache, high constant fever, malaise, anorexia, epistaxis and constipation. There had been a single attack of vomiting, profuse sweating and increasing apathy.

Examination.—Examination on admission showed a well developed, well nourished girl of 10 years, apathetic and slow to answer questions. The pupils were unequal; react to light; the eyegrounds showed evidence of increased intracranial pressure. The neck was held slightly rigid; there was some rigidity of extremities; the reflexes were active. The condition became slowly worse from day to day, until death Dec. 20, 1914.

Clinical Diagnosis.—Tuberculous meningitis.

Necropsy.—The body was that of an emaciated girl. The right lung contains numerous areas of bronchopneumonia. The bronchial lymph glands showed no tuberculous process. There was fatty degeneration of the liver. The stomach was negative. The duodenum as well as the ileum and the jejunum contained much bloody, tarry material. The duodenum contained a number of ulcers from pinhead to split-pea size.

Anatomic Diagnosis.—Tuberculous meningitis. Bronchopneumonia. Duodenal ulcers. Melena.

CASE 3.—A. J., a female child 2 years of age, was admitted to the hospital Jan. 2, 1915. Her father had pulmonary tuberculosis but "was cured." Birth was normal; her weight was 8 pounds. She was breast fed once each day and received cow's milk every four hours. When two weeks old she had convulsions, and five months before admission had bronchopneumonia. Since she was 6 months old had a discharging ear.

Duration of present illness, five days. She was fretful; coughed, vomited once and had fever. She appeared stupid. She had two or three stools daily, yellowish green and slimy.

Examination.—The examination on admission showed a dull, rather well nourished child, with cough, labored breathing, discharging left ear, carious teeth and enlarged, red tonsils. The lungs showed slight, not well defined dulness at right apex and axilla; moist râles over the entire chest. On the left side there were large mucous râles, over the upper right front and axilla and lower back, coarse râles; over the right posterior upper lobe there were subcrepitant râles and bronchial breathing. The stools were soft and yellow. No vomiting.

Death occurred Jan. 4, 1915.

Clinical Diagnosis.—Miliary tuberculosis.

Necropsy.—Well nourished girl. There are miliary tubercles in liver and spleen. An old fibroid nodule is seen in upper lobe of right lung. Right and left lower lobes are infiltrated. There are many typical undermined tuberculous ulcers opposite the mesentery in the ileum. Large healed tuberculous lymph gland at the beginning of jejunum. The duodenum is markedly reddened and shows several ulcers.

Anatomic Diagnosis.—Primary tuberculosis of upper lobe of right lung. Tuberculous lymph adenitis. Acute bronchopneumonia. Miliary tubercles of lungs, liver and spleen. Multiple tuberculous ulcers of small intestine. Multiple ulcers of duodenum.

CASE 4.—H. S., a female child 2½ years of age, was admitted to the hospital Jan. 7, 1915. Previous health had been good. Present illness was of two months' duration. Onset was with pains in back and right knee, nervous twitchings, cough and constipation.

Examination.—Examination on admission showed a well nourished child. Lungs: There was dulness at right apex front and back and slight impairment of note on left. Fine crackling râles were heard over the area of dulness. The extremities showed slight spasticity. Kernig's, Babinski's and Brudzinski's signs were positive. (The spinal fluid injected into a guinea-pig produced miliary tuberculosis.)

The condition grew worse and the patient died Jan. 12, 1915.

Clinical Diagnosis.—Miliary tuberculosis; tuberculous meningitis.

Necropsy.—Well nourished girl. The brain showed excess of fluid; the meninges were studded with small yellowish gray tubercles. A primary tuberculous lesion was found in upper lobe of right lung. There were miliary tubercles in spleen and liver. Duodenum was markedly reddened and its first portion contained numerous ulcers. Duodenum contained much blood.

Anatomic Diagnosis.—Primary tuberculosis of upper lobe of right lung. Tuberculous broncholymphadenitis. Miliary tuberculosis of spleen, liver and brain. Duodenal ulcers.

CASE 5.—A. S., a male infant 1 month of age, was admitted to the hospital Jan. 15, 1915. Birth normal, weight 9½ pounds. He was never breast fed. He was given condensed milk at first and later cow's milk. The present illness was of two weeks' duration; manifest loss of weight, coryza.

Examination.—Examination on admission showed an exceedingly emaciated, cyanotic, poorly cared for infant. There were excoriations of buttock and genital regions, and an ulcer on right foot. The stools passed contained a large mass of dark clotted blood. The infant died eight hours after entering the hospital.

Necropsy.—The body is that of a markedly emaciated male infant. Stomach contains mucus with brownish flecks. Duodenum: The surface is reddish and there is a slight amount of blood in the lumen. There are several ulcers of the peptic type, one the size of a split pea, the others pinhead in size.

Anatomic Diagnosis.—Atrophy. Multiple ulcers of duodenum.

Microscopic.—Section stained with hematoxylin and eosin. The section is of an ulcer overlying the pancreas, including a small portion of mucous membrane. The ulceration has extended completely through the duodenal wall and the greater portion of the base of the ulcer is formed by the pancreas. The portion of the mucosa seen in the section contains an excessive amount of connective tissue, in which lie large numbers of eosinophils. The remains of the muscular coats are only very slightly infiltrated with lymphoid cells, occasional small clumps are seen. The base of the ulcer over the pancreas consists of a necrotic zone of pancreatic tissue with practically no cellular infiltration. The eosinophils are present in excessive numbers in the connective tissue, although not as marked as in the mucosa.

Section stained for bacteria by the Gram-Weigert method. In the portion of the ulcer base consisting of partially necrotic gland tissue are a few pairs of diplococci. In the muscular part of the ulcer base nothing was found.

CASE 6.—B. L., a male infant, 3 months old, was admitted to the hospital Dec. 27, 1914. Present illness of three days' duration; fever and cough.

Examination.—Examination on admission showed a poorly developed, poorly nourished infant. Stools very loose, green and mucous; about four each day. He was started on a weak milk mixture and later given whey modified milk (Schloss). The stools continued loose and frequent, green and mucous throughout his entire stay in the hospital. Jan. 23, 1915, the stools contained dark and fresh blood. Death occurred early the following morning, January 24.

Necropsy.—The intestines are distended and of a bluish-green hue. Stomach normal. Duodenum: near the papilla is an ulcer measuring 5 mm. in diameter. The edges are smooth and the base is irregular and dark. There are several small pinhead sized ulcers in the first part of the duodenum. Ileum and colon are filled with black tarry fecal material.

Anatomic Diagnosis.—Atrophy. Duodenal ulcers. Melena.

Microscopic.—Section stained with hematoxylin and eosin. The section is taken through the base of the ulcer overlying the pancreas. No portion of the normal mucous membrane is present, only the wall of the ulcer involving the submucosa, muscular coats and pancreatic tissue. The base of the ulcer involving the duodenum is in part densely infiltrated with lymphocytes, in part practically free from cells. The connective tissue is, in part, of the young endothelial type, especially about a large blood vessel running parallel to the surface of the ulcer. In several places there are small areas of necrosis with fragmentation of the nuclei. In the pancreatic tissue there is a zone of demarcation between the surface of the ulcer and the normal pancreatic tissue in which there is a

coarse network of material which stains deeply with hematoxylin. Eosinphil cells are present in moderate numbers, especially about the vessels.

Section stained for bacteria by the Gram-Weigert method. The section shows only very few scattered diplococci in the base of the ulcer, in about the middle of the base and also slightly toward one side. None are to be found in the submucosa at either side of the ulcer.

CASE 7.—M. McB., a female infant, aged 5 months, was admitted to the hospital Jan. 29, 1915. The birth was normal, one of twins, weight 7 pounds, 6 ounces. Breast fed four weeks, supplemented by a cow's milk mixture. Then equal parts of cow's milk and water. The present illness was of indefinite duration. There had been gradual loss of weight.

Examination.—Examination on admission showed a poorly nourished infant with increased rate of respiration. Profuse discharge from nose and from both ears. The head was slightly retracted but no rigidity of neck. Chest: many medium and fine moist râles were heard over right back. Extremities were somewhat rigid.

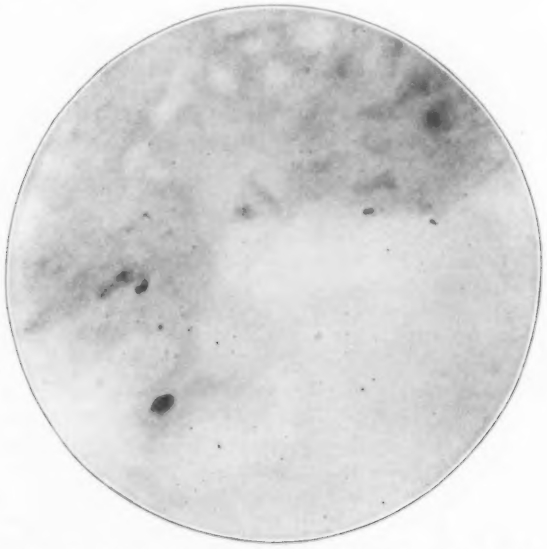

Fig. 2.—Photomicrograph of section from Case 7.

February 12, patient had a convulsion. Examination shortly afterward showed patient cyanotic, respiration shallow. Fontanel tense and slightly bulging. Right pupil was larger than left. There was no rigidity of neck. Chest: there was dulness over right back and front. High pitched medium and fine râles and tubular breathing. Lumbar puncture — cloudy fluid — and smears showed gram-negative bacilli.

Following a convulsion in the evening the patient vomited a small amount of "coffee ground" material which gave a positive test for blood.

The patient died the next morning, Feb. 13, 1915.

Clinical Diagnosis.—Influenzal meningitis and duodenal ulcer.

Necropsy.—No free fluid in the abdominal cavity. Intestines distended, of a bluish-gray color. Lungs: small area of consolidation in right upper lobe. Right ureter enlarged and swollen. Hemorrhage in left adrenal. Stomach negative. Duodenum: in the first one-third of the duodenum is an ulcer about ½ cm. in diameter with smooth and regular edges. No evidence of recent

hemorrhage. About ½ cm. below is another smaller ulcer. Small intestine and colon filled with tarry material.

The brain was covered with a thick, creamy exudate. Brain substance was soft and hyperemic.

Anatomic Diagnosis.—Purulent meningitis. Encephalitis. Duodenal ulcer, melena. Bronchpneumonia. Fatty degeneration of parenchymatous organs.

Microscopic.—Section stained with hematoxylin and eosin. The section of this ulcer is one of the most interesting of the series; it is the ulcer from which a *Streptococcus viridans* was cultivated which on injection into animals produced gastric and duodenal ulcers. This will be reported in detail by one of us at a later date. The ulcer is of further interest in that it shows a large vessel in its base just about ready to rupture. The tissues are well preserved, the mucous membrane to the side of the ulcer is practically intact; at the edge of the ulcer, the glands of Lieberkühn show some proliferation and tendency to form stellate cells in masses. The submucosa in the ulcer base is markedly edematous. This edema gradually subsides toward the normal tissue. The very edge is densely infiltrated with leukocytes. Where the inner muscular layer forms the base of the ulcer it is also edematous, especially so about the large vessel mentioned above, where it extends through to the serosa. The serosa is rather edematous in its entire extent under the ulcer. The wall of the vessel in the serosa is practically normal, the cells stain well and have a chromatin network; as it approaches the surface, however, there are no nuclei to be made out. A small lymph gland attached to the section shows considerable proliferation of the epithelioid cells in its germ centers.

Section stained for bacteria by the Gram-Weigert method: This section of the ulcer shows numerous diplococci in the base of the ulcer and in the side of the ulcer. At the level of the submucosa a short distance below the surface are a group of four pairs of diplococci, and scattered through the tissue adjacent there are other groups of diplococci. Figure 2 is a photomicrograph of this ulcer.

CASE 8.—R. R., a male infant 6 months of age, was admitted to the hospital Feb. 23, 1915. His birth was normal. He was breast fed only two months. The present illness began four days before admission, with convulsions, a bloody diarrhea, vomiting and high fever.

Examination.—Examination on admission showed a well nourished baby, quite ill, skin doughy, respiration rapid. There were a few fine râles over the chest. The abdomen was held rather rigidly, especially the right side where there was marked tenderness. No mass was palpable. Dark stools were passed.

Laparotomy was performed. The large intestines were greatly injected. Appendix was swollen, hyperemic, and the follicles prominent. The removed appendix showed no perforation. There was no intussusception. The dark contents of the ileum suggested blood. The patient vomited "coffee ground material." The death of the patient occurred about four hours after the operation, Feb. 23, 1915.

Necropsy.—The liver is large, firm and yellow, its section very fatty; the spleen pulpy and red and the kidneys swollen and yellow. The stomach contains a small amount of brown sticky mucus, but no ulcers. Duodenum: there is a large ulcer about 5 mm. in diameter. The surface of the duodenum is red and swollen. The colon shows many superficial ulcerations.

Anatomic Diagnosis.—Ulcer of the duodenum. Colitis. Fatty degeneration of parenchymatous organs. Acute splenic tumor.

Cultures made from ulcer base, at necropsy, were completely overgrown by colon bacilli, though some streptococci were present in the smears made from the original culture.

Microscopic.—Section stained with hematoxylin and eosin. The section is through one side and a portion of the base of the ulcer. There is relatively

little infiltration in the base and margin of the ulcer. There is a gradual slope from the margin to the bottom of the ulcer which in its middle position is made up of necrotic muscularis. Several large vessels in the submucosa near the surface of the ulcer show no evidence of thrombosis. The serosa is not infiltrated.

Section stained for bacteria by the Gram-Weigert method: The section shows very numerous streptococci throughout the entire base of the ulcer, at the surface as well as deep down in the tissue.

CASE 9.—J. M., a female infant 3 months of age, was admitted to the hospital March 18, 1915. The birth weight was 7½ pounds. She was never breast fed. The stools were always thin and greenish. The present illness was of four days' duration. The baby seemed ill. Examination on admission showed an extremely emaciated, small, weak baby. Respirations were shallow. There was impaired resonance over upper right front and back, medium râles were present over right back, a few over left. Abdomen scaphoid. Liver and spleen slightly enlarged. Death about eight hours after admission.

Necropsy.—The right upper lobe, paravertebral regions of both lower lobes are consolidated. The liver is very hyperemic. Stomach: Pyloric half of stomach is studded with numerous pinhead sized punched out ulcers. The same small pinhead sized ulcers are seen in the first part of the duodenum. The culture taken from the ulcer at necropsy showed only colon bacilli.

Anatomic Diagnosis.—Bronchopneumonia. Multiple ulcers of stomach and duodenum.

Microscopic.—Section stained with hematoxylin and eosin: The section shows the mucosa on either side of a small ulcer, which extends down to, but does not involve, the muscular layer. On one side the ulcer dips abruptly down, on the other side it slopes down gradually. The side of the ulcer, and to a lesser extent the base of the ulcer, are densely infiltrated with cells. The usual necrotic zone in the base is not evident. The Lieberkühn glands show no change.

Section stained for bacteria by the Gram-Weigert method. Streptococci were looked for in four sections. In only one were they found. Most typical was a pair of streptococci deep in the ulcer wall in the area of the submucosa. No other bacteria were found in the section.

CASE 10.—Charles P., a male infant 6 months of age, was admitted to the hospital April 14, 1915. The birth was normal. He was breast fed only three days, then given condensed milk. For two months before admission the baby had cried much, vomited after every feeding and was always constipated. Examination showed a small, poorly-nourished baby with shallow respirations. Loose, yellow, mucous stools. Death occurred on the evening of admission, April 14, 1915.

Necropsy.—Duodenum: There is an ulcer about 1 cm. from the pylorus, elliptical in shape, with blackened margins and roughened base, measuring ¾ by ½ cm. The remainder of the duodenum is very red, also the first part of the jejunum. The cultures from the ulcer base were overgrown with colon bacilli.

Anatomic Diagnosis.—Atrophy. Duodenal ulcer. Melena.

Microscopic.—Section stained with hematoxylin and eosin: The section shows an ulcer of moderate size, extending down into the submucosa. There is marked infiltration of the base as well as of the sides of the ulcers. One side of the ulcer is abrupt; the other side slopes gradually down from the normal mucosa. The muscular layers show no infiltration.

Section stained for bacteria by the Gram-Weigert method: In the base of the ulcer just below the surface there is a pair of streptococci. No other bacteria to be seen in the section.

CASE 11.—A. S., a male infant 2 months of age, was admitted Aug. 9, 1914. His birth weight was 7 pounds. He was being fed cow's milk, one-third dilution. For two days he had fever and diarrhea; green stools every hour, but no blood. There was no vomiting. Examination on admission showed a small fairly well nourished infant. There was a fine papulovesicular rash over the entire body. Abdominal walls were normally resistant. Liver was slightly enlarged. There was a trace of albumin and a few leukocytes in the urine. The stools were frequent, green and mucous. The infant was given albumin milk. For a short time there was a little improvement, but stools were rather frequent, green and mucous and there was some regurgitation. September 16 there was a sudden rise of temperature to 103 F. It then dropped below normal. Stools began to show traces of blood. Patient vomited bright red blood and later "coffee ground" material. No bleeding points in mouth or pharynx.

The next day, September 17, patient appeared in better condition. There was no more regurgitation of food or blood. Stools gave positive chemical test for blood.

September 19, patient appeared paler, had no rigidity of abdomen, but edema of dorsal surface of hands and feet. September 21 there was edema of face and head; also there was regurgitation of food, but no blood. Abdomen was rather tense. Fulness in gastric area was marked but no peristaltic waves were seen. September 22 there was general edema, dyspnea and continuous regurgitation. Death Sept. 24, 1914.

Clinical Diagnosis.—Gastro-enteritis. Duodenal ulcer. Acute nephritis.

Necropsy.—Moderate state of nutrition. The kidney cortex is red and swollen. Stomach is markedly reddened, no ulceration. In the first portion of duodenum there is a small punched out area with small central scar.

Anatomic Diagnosis.—Acute nephritis. Healed duodenal ulcer.

Microscopic.—Section stained with hematoxylin and eosin: The block was taken through the middle of the little depression which is all that is to be seen of the ulcer at the necropsy. The section shows a swelling instead of a depression in its middle portion. This swelling is due to a very marked infiltration with small round cells, of all of the coats of the duodenal wall. The mucosa lining over the swollen portion is not intact. The most marked infiltration is in the layer of the Lieberkühn glands. The glands are forced wide apart by the lymphocytic infiltration. The inner muscular layer is so separated by the infiltration that it is difficult to make it out distinctly. The outer muscular coat is also densely infiltrated, and along the vessels passing through there is a marked collection of cells. The serosa is infiltrated for a considerable distance beyond the thickened area.

Section stained for bacteria by the Gram-Weigert method: In the middle of the thickened area where the mucosa and submucosa are markedly infiltrated, there are numerous diplococci lying in the tissues a short distance below the surface. In this same area there are several chains of four members.

Before passing on to a discussion of these cases, it seems advisable to give in detail the results of an examination of all the ulcers still available for microscopic study of Series I and II previously reported by one of us.[1, 5] In order not to take up too much space the ulcers have been numbered, and with each one the case number and name as well as the series is given, so that they can be identified in the previous communications.[1, 5]

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5. Helmholz, H. F.: Duodenal Ulcer in Infancy, Arch. Pediat., 1909, xxvi, 661.

The sixteen cases reported were practically all cases of atrophy, with single or multiple duodenal ulcers. Of the sixteen cases described there were 14 ulcers from 11 different cases available for study. No blocks of tissue were available, only sections stained with hematoxylin and eosin. In some instances only a single section was at hand. The sections were decolorized with acid alcohol and were stained by the Gram-Weigert method.

The findings in the ulcers were as follows:

Ulcer 1 (Case 1, Series I, Ernest M.).—Section of the ulcer shows numerous streptococci in tissue just beneath ulcer surface. The cocci are arranged in a row parallel to the surface in four groups of twos.

Ulcer 2 (Case 2, Series I, Hildegrad O.).—After prolonged search it was impossible to find any streptococci. Two sections were available.

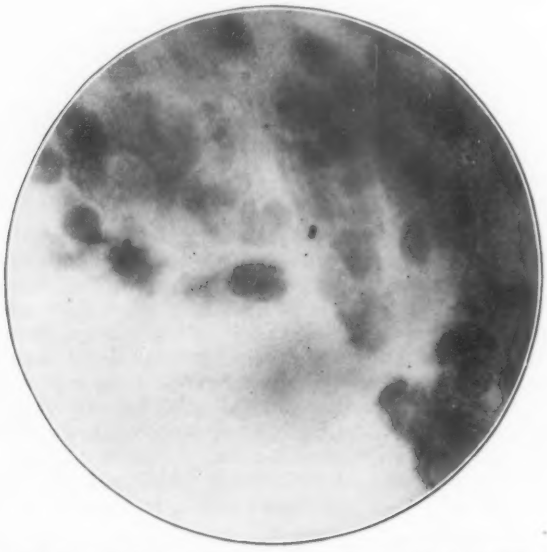

Fig. 3.—Photomicrograph of section in Case 8, Series I, Timm M., Ulcer 8.

Ulcer 3 (Case 4, Series I, Willie R.).—Only single partially healed ulcer was available, repeated prolonged search failed to reveal any streptococci.

Ulcer 4 (Case 5, Series I, Kurt Kruggel).—The section is of an ulcer that perforated the duodenal wall. In the portion of the ulcer where the base is formed by the inner muscular coat there is seen just below the surface of the ulcer a pair of streptococci somewhat larger than usual. No other bacteria were found in the entire section.

Ulcer 5 (Case 6, Series I, Alfred K.).—Two sections of an ulcer that differs from the others in that it shows very much more leukocytic infiltration than any of the other ulcers. Repeated prolonged search did not reveal any bacteria.

Ulcer 6 (Case 7, Series I, Erich S.).—Two sections of the ulcer were at hand. Careful search on several occasions revealed no bacteria whatsoever in the sections.

Ulcer 7 (Case 8, Series I, Timm M.).—A small ulcer extends down into the muscular coat with the remains of a small clot in the ulcer base. In the base of the ulcer just beneath the surface, where it is made up of the muscular layer, there are three pairs of diplococci.

Ulcer 8 (Case 8, Series I, Timm M.).—Section is through the wall of an ulcer that has completely eroded the duodenal wall. In the wall consisting of submucosa there are three distinct pairs of diplococci to be seen just below the surface of the ulcer. No bacteria were found elsewhere in the section. A pair of the cocci are shown in Figure 3.

Ulcer 9 (Case 4, Series II, H. Ulrich).—Ulcer is a rather superficial one in which only a portion of the ulcer extends deeply down into the muscular coat. On the surface towards one side lying deep in the tissue are two groups of diplococci. The rest of the section shows no bacteria.

Ulcer 10 (Case 4, Series II, H. Ulrich).—The ulcer is partially healed, epithelium growing in from the side. In almost the middle of the ulcer, just

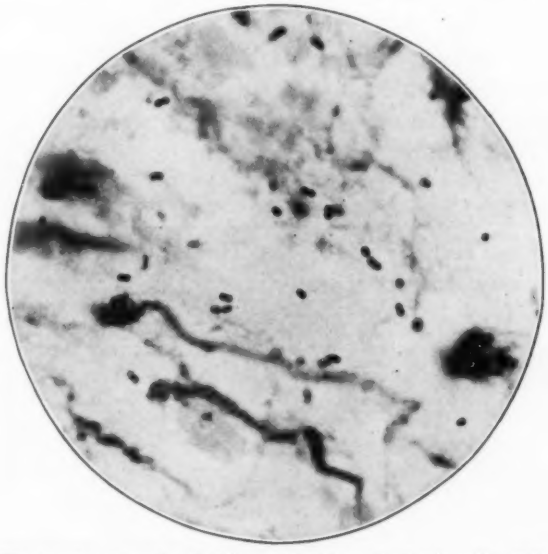

Fig. 4.—Photomicrograph of Ulcer 12, Case 5, Series II, Alf. Gerlach.

below surface, there is a group of four pairs of diplococci and at a short distance another pair of diplococci.

Ulcer 11 (Case 4, Series II, H. Ulrich).—Sections contain two ulcers. In base of the larger one are seen numerous cocci lying in the tissue just beneath the surface of the ulcer. The cocci are in short chains and pairs.

Ulcer 12 (Case 5, Series II, Alf. Gerlach).—This ulcer is interesting because of the fact that it has absolutely no inflammatory reaction about it and is the type of ulcer that is usually considered an erosion rather than a definite ulcer. This ulcer is of all the ulcers the one in which the streptococci were most numerous. Practically the entire base of the ulcer, made up of submucosa, is studded with diplococci and streptococci in short chains. This portion of the section is shown in Figure 4.

Ulcer 13 (Case 5, Series II, Alf. Gerlach).—Another section of the above case contains two ulcers both of which show in their base and sides numerous diplococci and streptococci.

ULCER 14 (Case 6, Series II, K. Lieback).—Stained with hematoxylin and eosin. The section could not be restained because of a broken slide. The ulcer shows, however, in the portion of the base consisting of submucosa, a single pair of streptococci of very definite outline.

SUMMARY

In ten out of fourteen ulcers from the two previously reported series, in which only one or two sections of each were available for study, diplococci were found in the ulcer base, and in all eight ulcers of our present series diplococci were found in the ulcer in such numbers and in such a position that they presumably are of etiologic significance. Furthermore, in one of the cases a *Streptococcus viridans* was isolated from the duodenal ulcer, which on injection into rabbits and dogs localized in the pyloric end of the stomach and duodenum and there produced hemorrhages and ulcerations. This case was the only one in which we were able to obtain the organisms in pure culture, and the only one in which the necropsy was performed shortly after death. In two other cases a few streptococci were seen in the bouillon cultures, but the cultures were so overgrown with colon bacilli that it was impossible to isolate the streptococci. In the fourth case only the colon bacillus was grown.

Rosenow[4] has recently shown that from duodenal and gastric ulcers removed at operation, a streptococcus viridans can be constantly isolated, which when injected intravenously into dogs and rabbits will tend to localize in the stomach and duodenum and there produce first hemorrhage and later ulcers. In explanation of this phenomenon he assumes that the streptococcus producing duodenal ulcer finds in the duodenum conditions peculiarly favorable for its development, and therefore tends to localize there when injected intravenously into animals. Whether this tendency to localize is a question of oxygen tension or of some other factor does not interest us at the present time; only the fact that there is a streptococcus which shows this peculiar predilection for the duodenal mucosa. In other words, we can assume that at times, because of circumstances at present not well understood, the streptococcus takes on characteristics which make it tend to localize in the duodenum when infecting an infant. We have thus a working hypothesis which explains very readily the epidemic appearance of duodenal ulcers; namely, that we have an infection with a very definite organism, which, carried to the infant either through the digestive tract or through the air passages, tends to localize in the duodenum. This same tendency to localize in specific tissue has been shown by Rosenow to be true of the streptococcus isolated from cases of rheumatic fever, endocarditis, appendicitis and herpes zoster, when injected into animals. Not only is the organism specific when isolated

from the characteristic lesion, but usually when isolated from the portal of entry, such as the tonsils, an alveolar abscess, etc. Rosenow has quite conclusively established the fact that gastric and duodenal ulcer of the adult are the result of an infection with a streptococcus of particular virulence. That this holds good for duodenal ulcers of the infant also is very definitely shown by the following facts summarizing our work:

1. The appearance of duodenal ulcer in epidemic form.

2. The presence of diplococci and streptococci in all the ulcers of our present series available for study, and in ten out of fourteen ulcers of a previous series of cases.

3. The isolation, at necropsy, from one ulcer, of a *Streptococcus viridans* which when injected into dogs and rabbits localized in the pyloric end of the stomach and the duodenum and produced there hemorrhages and ulcers.

Regarding the symptomatology of duodenal ulcer we have nothing to add, it being practically never possible to diagnose the condition until there are complications from the ulcerative process, such as rupture of a vessel, or perforation into the peritoneal cavity.

Death occurred within forty-eight hours in all cases of hemorrhage, excepting one in which death occurred after one week from an intercurrent infection; the ulcer was practically healed, showing how rapid the healing is even in infants who are markedly atrophic. Occurring as a complication usually in atrophic infants, the prognosis is always bad. Transfusion as described by one of us[6] might be of some value if performed immediately.

In conclusion we wish to thank Dr. Rosenow for his kind suggestions and his assistance in this work.

6. Helmholz, H. F.: The Longitudinal Sinuses as a Place of Preference for Transfusion in Infancy, Am. Jour. Dis. Child., 1915, x, 147.

A COMFORTABLE ARRANGEMENT FOR THE SEPARATE COLLECTION OF URINE AND FECES OF MALE INFANTS *

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Of the utmost importance in the quantitative investigation of infant excretions is the absolutely complete collection of the separate urine and feces for the desired period. Hardly second in importance is the question of comfort for the child, for it is possible that a constrained or unusual posture appreciably affects the metabolism. For example, one arrangement, in which the body is partly supported as in a hammock, brings considerable pressure on the lower part of the back and in some cases has seemed to cause increased peristalsis. With another method the bed is necessarily slightly tilted and the child held from slipping by an elaborate system of straps so arranged as not to press on any part of the body but holding shoulders and arms in an almost immovable position.

The arrangement here described is recommended as combining accuracy of collection with a maximum of comfort for the child. It is mainly a satisfactory combination of details from methods in use. The Finkelstein[1] rubber and glass contrivance, which experience in the Babies' Hospital has shown to be the most accurate for collecting urine, is somewhat modified to suit an entirely different arrangement for supporting the child. The means used to hold the child from slipping, although adopted independently here, is practically the same as that employed in Talbot's[2] metabolism bed. An air cushion is used in Schabad's[3] scheme, but so far as is known no use has elsewhere been made of the air cushion with an opening in the rim. This method can be carried out on any crib or bed by making the necessary modifications of the mattresses. It is more convenient to use an adjustable crib in which the bottom can be raised to bring the child to a convenient level for handling.

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* From the Laboratories of the Rockefeller Institute for Medical Research and the Babies' Hospital.

* Thanks are due to Dr. E. A. Morgan, Resident Physician of the Babies' Hospital, for taking photographs.

1. Bendix and Finkelstein: Deutsche med. Wchnschr., 1900, xlii, 672.

2. Talbot, F. B.: Apparatus for Metabolism Experiments in Male Infants, Jour. Am. Med. Assn., Nov. 27, 1908, p. 1818.

3. Schabad: Arch. f. Kinderh., 1908, xlviii, 402.

The following articles are required:

1. Mattresses, all of the ordinary thickness. One piece (*A*) 20 x 23 inches; two pieces (*B*) 6 x 8 inches; two pieces (*C*) 8 x 16 inches, for a crib 21 x 48 inches.

2. Rubber air cushion. Outside diameter 12 inches; inside diameter 3½ inches. Opening when inflated about 2 inches.

3. Cloth cover for cushion, consisting of two 18-inch squares with a circle 3½ inches in diameter cut out of the center, the pieces being stitched together around the circle and free on all sides.

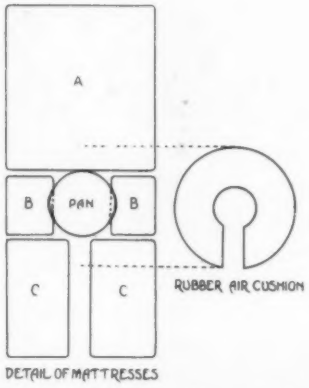

Fig. 1.—Diagram showing detail of mattress and rubber air cushion.

4. Enamel ware pan with flange, 9 inches in diameter.

5. Ordinary male glass urinal.

6. Finkelstein rubber attachment.

7. Glass tube (after Finkelstein, but lighter and differently bent).

8. Cloth strap made of two pieces of webbing, each 6 inches wide, one 28 inches long, placed on the other which is 36 inches long, and the two stitched together across the middle.

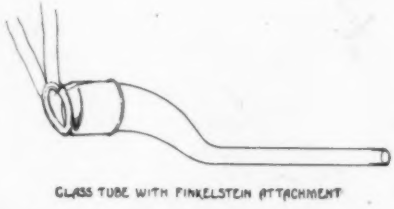

Fig. 2.—Drawing of glass tube with Finkelstein attachment.

Directions for setting up:

Cover mattresses separately with sheets, pinning on under side.

Place mattresses and pan in position according to diagram Figure 1.

It is convenient to lay a circle of oiled silk in the bottom of the pan.

Inflate rubber cushion not too hard, place within cloth cover, and pin down snugly on mattresses, with opening toward bottom of bed.

Fig. 3.—Material of metabolism bed assembled.

Fig. 4.—Infant in the metabolism bed with apparatus applied.

Place cloth band across mattress A with lower edge just above lower edge of mattress and pin ends of bottom strap tightly to sides of mattress.

Place child on bed with anus directly over hole in rubber cushion, buttocks resting comfortably on cushion. If necessary, lay a pad under back above where he rests on the cushion.

Insert the large end of the glass tube (Fig. 2) in the end of the rubber attachment, pushing it in well.

Place small end of glass tube in neck of urinal and make the latter secure between cushions C.

Attach the rubber to the child, putting scrotum and penis within the large end of the glass tube and bring the rubber close to the body. Draw the rubber straps around the body, crossing on the back and fasten ends together in front with adhesive plaster. Make secure but not tight. Place absorbent cotton under rubber wherever it seems advisable.

Pin the ends of the upper strap of the cloth band snugly around the body.

The infant's legs rest on cushions C.

Ankles should be tied loosely by wide tapes, with absorbent cotton under the tape, to the sides of the crib, allowing some movement of the legs but not enough to cause disturbance of the urinal.

The glass tube in the rubber attachment resting in the space between the ends of the air cushion gives sufficient drop to assure the flow of the urine into the urinal.

Cushion B can easily be removed to facilitate changing the enamel pan.

A thin board between springs and mattresses will prevent any air currents from reaching the child from beneath.

With a very small or very restless child frequent inspection at the beginning is advisable to make sure that the rubber adjustment has not slipped, but ordinarily the necessary visits at intervals to remove the samples of urine and feces to the icebox are sufficient to make sure that all is right.

OXALIC ACID EXCRETION IN THE URINE OF CHILDREN*

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So far as I have been able to find, there are but two references in pediatric literature to the determination of the oxalic acid content of the urine of infants. The first is in the handbook of Pfaundler and Schlossmann[1] published in 1906, and is as follows: "The studies of Sedgwick at the Heubner Clinic have shown us that children excrete absolutely larger amounts of oxalic acid than adults."

The second reference appeared last year in an article by Hans Aaron and Marianne Franz,[2] on the "Organic Acids in the Urine of Infants." They state that, so far as they know, no examinations of infants' urines for oxalic acid appear in the literature. They mention records in the literature of the finding of 9 to 11 mg. of oxalic acid in the day's urine of a dog during the second week of starvation, and state that this must be endogenous oxalic acid. In addition to this, it is known that oxalic acid may be formed in the intestine as a byproduct of carbohydrate fermentation. From these facts and other studies on adults, it is generally accepted that there may be an endogenous formation of oxalic acid in the human metabolism, even on an oxalic acid free diet. It is also recognized, from examinations of the urine of rabbits, that a carbohydrate diet may increase the excretion of oxalic acid in the urine. This is supposed to be due to the imperfect oxidation of the glycuronic acid produced and a secondary formation of oxalic acid.

Hildebrandt[3] gave rabbits that were being fed oats 30 gm. of glucose per day. These animals died with symptoms of acute intoxication. However, when he gave such animals lime with the same amount of sugar, they bore the diet without inconvenience. He explained this protective action of calcium by the supposition that the oxalic acid formed was bound by the calcium and rendered nontoxic. The work of Aron and Franz led to the following conclusions:

1. The urine of infants contains small amounts of oxalic acid almost without exception. It is found even when they are on oxalic acid free diets, such

* Submitted for publication Sept. 6, 1915.

1. Pfaundler and Schlossmann: Handbuch der Kinderheilkunde, 1906, ii, 522.
2. Aaron and Franz: Organic Acids in the Urine of Infants, Monatschr. f. Kinderh., 1914, xii, O., p. 645.
3. Hildebrandt, H.: Ztschr. f. physiol. Chem., xxxv, 141.

as breast milk or cow's milk and sugar, and this must be considered as either from the intestine, as a byproduct of carbohydrate fermentation or formed in the intermediary metabolism, that is, endogenous oxalic acid formation.

2. After feeding of cane sugar or dextrin-maltose preparations, the oxalic acid excretion was somewhat higher than on an exclusively milk diet. Starchy additions to the diet caused a definite increase in oxalic acid excretion.

3. No increase of the oxalic acid excretion could be demonstrated in cases of acute feeding disturbances.

METHODS

One reason that this subject has been so long neglected is that the methods of determination of oxalic acid in urine are so tedious and unsatisfactory.

This work was begun in 1905 at the suggestion of Professor Leo Langstein. The Autenrieth and Barth[4] method was used for the work done then. In this method the oxalic acid is thrown down as calcium oxalate; the oxalic acid is then isolated by heating with hydrochloric acid and dissolving in ether. An aqueous solution of the oxalic acid is finally obtained and reprecipitated with calcium chlorid. The precipitate is ignited and weighed as calcium oxid. The results were not published, as I found that the final precipitates contained phosphates.

The first part of the work presented here was done by the Salkowski method. In this method the urine is concentrated on the water-bath to one-third of its original volume, treated with concentrated hydrochloric acid, and the oxalic acid then dissolved in ether by shaking. As will be seen from the tables, this method gives lower results than the one devised by Albahary.[5] This is largely due to the difficulty of extracting all of the oxalic acid by shaking with ether. Although Salkowski advises shaking four times with ether, I found that the seventh wash ether still contained oxalic acid, and Albahary quotes Luzatto as saying that the eighteenth extraction still contained oxalic acid.

The Albahary method consists in adding sodium carbonate to the urine, and concentrating it to one-third of the original volume over the water-bath. This alkalinization before heating has the advantage of preventing the volatilization as pointed out to us by Dr. Hall, or decomposition, of the oxalic acid which is so prone to occur when an acid solution of oxalic acid is heated. This concentrated solution is then treated with magnesium chlorid and ammonium chlorid and filtered to remove the phosphates. The filtrate is treated with calcium chlorid and acetic acid. The precipitate is removed and ignited. The amount of oxalic acid is then reckoned from the calcium oxid obtained. This method is much shorter and control experiments with definite amounts of oxalic acid show that more, though not all, is recovered by this procedure.

As there are no reported results of oxalic acid determinations in the urine of the new-born, they are shown in Table 1.

The results in Table 1 were obtained by the Salkowski method. The results shown in Table 2 were obtained by the Albahary method and are considerably higher.

If the results by the Salkowski method are averaged, we find a daily excretion of 2.75 mg., and by the Albahary method, 4.78 mg.

These were all normal breast fed new-born infants. The oxalic acid must therefore be endogenous. The quantities are distinctly larger than those found for older infants by Aaron and Franz.

Baby Ols. when 4 months old showed by the Salkowski method 7.19 and 4.77 mg. per day oxalic acid excretion.

4. Autenrieth and Barth: Ztschr. f. physiol. Chem., 1902, xxxv, 327.
5. Albahary, M.: Compt. rend. Acad. d. sc., 1903, cxxxvi, 1681.

Harold R., 4 years old, with spasmophilia, gave a daily average by the Salkowski method from seventeen specimens, covering a period from October 31 to January 31, of 9.3 mg. Clifford Knick, 6 years old, with acute enteritis, enlarged tonsils, and adenoids, averaged 11.42 mg. for a period of eight days, also by Salkowski's method.

Violet Wall., aged 7 years, averaged by Salkowski's method, 10.53 for seven days during January. On January 10, she excreted 20.44 mg. On January 20 she was etherized for a tonsillectomy and the next day she excreted 0.45 mg. only, coming back to 13.25 on the following day.

TABLE 1.—Oxalic Acid Determinations in the Urine of the New-Born

Infant	Time Collected	Quantity c.c.	Oxalic Acid mg.
Sim.	First four days after birth...	146.5	3.59
Ols.	Second to fifth day after birth	248.5	4.94
Byr.	First four days after birth...	140.0	17.30
Hor.	Seventh day after birth.....	205.0	9.43
Hor.	Eighth day after birth.....	265.0	3.18

TABLE 2.—Oxalic Acid Determinations by Albahary Method

Infant	Time Collected or Age	Quantity c.c.	Oxalic Acid mg.
Byr.	Fifth, sixth, seventh days after birth	307.0	15.50
Byr.	Eighth and ninth days.....	239.0	11.23
Shaw	Three days	168.0	13.47
Loog.	First seven days after birth..	334.0	12.35
Hay	Two days	89.0	18.01
Hay	Two days	39.0	12.35
Hay	Four days	34.0	23.81
Gusta	Four days	76.0	24.48
Reine	Five days	123.0	22.01

Dorothy Rub., aged 6 years, with bronchitis and adenoids, showed an average of 9.9 mg. for six days by the Salkowski method. Hilding Berg., 11 years old, with chronic cardiac valvular disease and rheumatic fever, gave the low average of 3.04 mg. for four days by the Salkowski method, and on one of these days no oxalic acid could be demonstrated in the urine. In the older children, also, the results by the Albahary method were definitely higher. Doris Russel, 6 years old, showed an average daily excretion of 21.6 mg. for six days. D. Sten., 2 years old, with a healed pleurisy, by the Albahary method, for a preliminary period of fourteen days, on a general diet, gave a daily oxalic excretion of 21.77 mg. For the next four days on an oxalic acid free diet the daily excretion averaged 16.42 mg. The last day of this period she excreted 5.62 mg. only. During the following four days she was given 50 c.c. of rhubarb sauce daily and her oxalic acid excretion increased to an average of 32.85 mg. per day. The amount of oxalic acid in rhubarb is given by Neuberg[6] as 2.4 gm. per kilogram of substance.

6. Neuberg, Carl: Von Noorden's Handbuch der Pathologie des Stoffwechsels, 1907, ii, 490.

SUMMARY

The older methods of determination of oxalic acid are tedious and imperfect. The Albahary method gives better results and is much more rapid.

New-born infants excrete oxalic acid in the urine in varying amounts up to 9 mg. per day.

Older children excrete oxalic acid in considerable quantity, and one child, fed on rhubarb, showed a definite increase in oxalic acid excretion during the period of rhubarb feeding.

If we accept the usual figures which are given for the oxalic acid excretion in adults, given by Neuberg as from 15 to 20 mg., the excretion in children is relatively and at times absolutely higher.

THE PHTHALEIN TEST IN ORTHOSTATIC ALBUMINURIA *

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ST. LOUIS

Orthostatic albuminuria is a comparatively common condition, especially in children, and has been the subject of frequent investigation, yet the pathology of the condition is far from being clearly understood. Although it is not commonly classified among the nephritides, actual opportunities for histologic examination of kidneys from individuals with orthostatic albuminuria, have been very infrequent, chiefly because of the benign character of the affection. Such opportunities for necropsy examination as have occurred have failed to reveal any of the usual pathologic findings of a nephritis. Until the accumulation of necropsy findings or physiologic experimentation shall have cleared up the pathogenesis of the condition, we must rely chiefly on clinical observation and renal tests for our knowledge of the factors involved in its production.

In a clinical investigation, the question of the integrity of the kidney function is naturally one of the first to suggest itself. A few earlier studies of kidney function in orthostatic albuminuria, using salt, potassium iodid, or indigo-carmin excretion, etc., as tests, have been made, but the results were indefinite and inconclusive. So far as I know, no tests of renal function have been made with the more delicate methods introduced in recent years, and hence this study was undertaken. In a preliminary note[1] I reported briefly the results of the phenolsulphonephthalein test in four cases, which are included in this paper.

Only marked cases of the so-called lordotic type were used in these studies—cases in which large amounts of albumin appeared in the urine after such a simple procedure as having the child stand against the wall for ten minutes with the heels and back of the head touching the wall, thus throwing the shoulders well back, and bringing the lumbar spine forward. In all these cases the urine was free from albumin after a night's rest in bed. Children with slight or moderate degrees of orthostatic albuminuria were not used for tests. In all

* Submitted for publication Sept. 18, 1915.

* From the Department of Pediatrics, Washington University, and the St. Louis Children's Hospital.

1. Read before the Washington University Medical Society, May 11, 1914; reported in the Journal of the Missouri Med. Soc., June, 1914, and the Bulletin of the Washington University Med. School, June, 1915.

cases the albumin precipitated on the addition of acetic acid, although this precipitate was usually increased by heating. No casts were found in the urine.

The manner of making the tests was as follows: The cases were referred from among the children coming to the out-patient dispensary and admitted to the hospital for the carrying out of the tests. On admission, the children were kept in bed over night and the morning urine examined for albumin. If free, the child was placed in the position described above for ten or fifteen minutes, and the urine examined again. Cases then showing large amounts of albumin (enough to give a curdy precipitate with the acetic acid test), were utilized. The patient was again put to bed and the following day—the child meanwhile not being allowed up—a phenolsulphonephthalein test was made with the child in the position of accentuated lordosis throughout the whole time of the test. In the test 0.6 gm. of phenolsulphonephthalein contained in 1 c.c. of solution, was injected into the muscles of the back, as advised by Rowntree and Geraghty. The urine excreted in the first and second hour after injection was collected separately and the amount of phenolsulphonephthalein present in each estimated by means of a Du Boscq colorimeter. The children were then allowed to play about the ward for two or three days, an interval of time sufficient to get rid of the phthalein used in the first injection. On the third or fourth day, after the child had been in bed over night, the test was repeated with the child in the normal position, that is, lying flat in bed, not accentuating the lumbar curve. In some of the cases, the time of first appearance of the drug in the urine was noted, but not in all. The position of accentuated lordosis was maintained during the two hours required for the test by having the child lie on its back with several hard pillows under the lumbar spine, or by sitting in a chair, with a stick across the back, held in the crooks of the elbows. In three instances the order of the tests was reversed, the test in the normal position being made first, and in one case a longer interval elapsed between tests. The protocols are as follows:

PROTOCOLS

CASE 1.—Richard P. (No. 3828), aged 12 years, came to the dispensary in January, 1913, because of enuresis. He presented the typical findings of an orthostatic albuminuria, all other physical examination being negative except for an accidental heart murmur, which disappeared during the period of observation. The first phthalein test was made Feb. 19, 1913, with the patient in bed.[2] The first appearance of the drug was in five minutes, 62.8 per cent. being excreted in the first hour, and 5 per cent. in the second, or 67.8 per cent.

2. It will be remembered that the drug normally appears in the urine in from five to eleven minutes after intramuscular injection, 40 to 65 per cent. being excreted in the first hour after the initial appearance, and 65 to 80 per cent. in two hours.

in two hours. The test Feb. 22, 1913, with the patient in the position of accentuated lordosis gave the first appearance in eight minutes, with 42.8 per cent. excreted in the first hour and 10 per cent. in the second, or 52.8 per cent. in the two hours; that is, 20 per cent. less in the first hour and 15 per cent. less in the two hours, than when he was in the normal position. April 11, 1914, the test was repeated, with the patient in the normal position, when 59 per cent. was excreted in the first hour and 5 per cent. in the second, or 64 per cent. in the two hours — a normal output, and no evidences of nephritis a year after the first tests.

CASE 2.—Blanche C. (No. 3302), 11 years old, came to the dispensary because of enuresis. Typical orthostatic albuminuria with no other abnormality. First phthalein test on May 4, 1914, with the patient in the position of accentuated lordosis. Time of appearance not noted. First hour, 39 per cent. excreted, second hour, 20 per cent., or 59 per cent. in two hours. Second test on May 5, 1914, with patient in the normal position. First hour, 48.8 per cent. excreted; second hour 10 per cent., or 58.8 per cent. in the two hours; that is, in the lordotic position 10.8 per cent. less in the first hour, but an equivalent amount in the two hours.

CASE 3.—Robert M. (No. 5027), 9 years old, came to the dispensary Feb. 18, 1914, because of enuresis and trachoma. Physical examination reveals no other abnormalities except an orthostatic albuminuria. Phthalein test March 11, 1914, in position of accentuated lordosis, showed first appearance in eight minutes, with 35 per cent. excreted in the first hour and 5 per cent. in the second, or 40 per cent. in the two hours. Phthalein test May 6, 1914, normal position, showed 66⅔ per cent. excreted in the first hour, 20 per cent. in the second, or 86⅔ per cent. in the two hours; that is, in the lordotic position, over 30 per cent. less for the first hour and 46 per cent. less for the two hours.

CASE 4.—Theodore K. (No. 3216), 10 years, came to the dispensary in May, 1910, because of hypertrophied tonsils and adenoids, with enuresis. He had, and still has, a typical orthostatic albuminuria, which has been observed now for over five years, with no further evidences of a nephritis. Subjected to the phthalein test May 2, 1914, in the position of accentuated lordosis. First hour, 41.2 per cent. excreted and in the second hour 10 per cent., or 51.2 per cent. for the two hours. Test repeated May 6, 1914, with boy in normal position, when 48.3 per cent. was excreted in the first hour, and 15 per cent. in the second, or 63.3 per cent. in the two hours. This case, therefore, shows approximately 12 per cent. less excreted for the two hours, and 7 per cent. less the first hour while in the lordotic position.

CASE 5.—John M. (No. 8192), aged 10 years, came to the dispensary because of urticaria and adenoids. No other abnormality except an orthostatic albuminuria. First phthalein test March 26, 1915, with boy in normal position. Time of appearance eight minutes. First hour, 55 per cent. excreted; second hour, 15 per cent. excreted, or 70 per cent. in two hours. Second phthalein test March 31, 1915, lordotic position. Time of appearance, eleven minutes. First hour, 30 per cent. excreted; second hour 40 per cent. excreted, or 70 per cent. in the two hours. In the lordotic position, 25 per cent. less was excreted during the first hour, but an equal amount for the two hours.

CASE 6.—Francis M. (No. 8317), aged 8 years, came to the dispensary because of an aphthous stomatitis. Typical orthostatic albuminuria in addition. First phthalein test April 16, 1915, in the lordotic position. Time of appearance, eight minutes. First hour 45 per cent. excreted, and second hour 15 per cent. excreted, or 60 per cent. in the two hours. Second phthalein test April 19, 1915, with boy in the normal position. First appearance, six minutes. First hour 60 per cent. excreted, second hour 10 per cent., or 70 per cent. excreted in the two hours. There was, therefore, 15 per cent. less excreted in the first hour and 10 per cent. less in two hours while in the lordotic position.

CASE 7.—Rosie T. (No. 8406), aged 11 years, came to the dispensary for bronchitis and orthostatic albuminuria. First phthalein test May 1, 1915, in normal position, showed 55 per cent. of the drug excreted in the first hour, and 12 per cent. in the second, or 67 per cent. in two hours. Second test on May 3, 1915, in the lordotic position, with 40 per cent. of the drug excreted in the first hour and 20 per cent. in the second, or 60 per cent. in the two hours. There was, therefore, 15 per cent. less excreted during the first hour in the lordotic position, and 7 per cent. less in the two hours.

CONTROL 1.—Frank R. (No. 3910), normal, well boy, subjected to the phthalein test Feb. 19, 1913, in position of accentuated lordosis and showed 56.5 per cent. excreted in the first hour and 15 per cent. in the second, or 71.5 per cent. in the two hours. Test repeated Feb. 22, 1913, with patient in the normal position, when 55 per cent. was excreted in the first hour and 15 per cent. in the second, or 70 per cent. in the two hours — approximately the same result in both positions.

CONTROL 2.—Alvin J. (No. 5572), aged 7 years, up and about the wards, with no urinary abnormality whatever. Subjected to the phthalein test in the position of accentuated lordosis May 6, 1914, and showed 76.8 per cent. of the drug excreted in the first seventy minutes (was unable to void at sixty minutes) and 5 per cent. in the next fifty minutes, or 81.8 per cent. in two hours. Test repeated with the boy in the normal position May 8, 1914, when 71.3 per cent. was excreted in the first hour and 12.5 per cent. in the second, or 83.8 per cent. in the two hours; that is, approximately the same for the two-hour intervals, but 5.5 per cent. more in seventy minutes, lordotic position than in one hour, normal position.

The accompanying table summarizes the results of the seven cases and controls.

It will first be noticed that all of the cases except Case 2, came within the limits of the normal as regards phthalein output when in the normal position. Case 2 is slightly under the average, but hardly enough to call the phthalein output abnormal. The seven cases averaged 69 per cent. output in two hours and 56.6 per cent. in the first hour. It was found, however, that when the child with orthostatic albuminuria maintained the lordotic position during the test, there was apparently a slowing up of the excretion of phthalein. This was most marked in the first hour, when the average for the seven cases was 39 per cent., in contrast to 56.6 per cent. in the normal position—a decrease averaging 17.6 per cent. The total difference for the two hours is less, averaging but 12.9 per cent. Moreover, it will be noted that in every instance this retardation occurs in the first hour, the extremes being 7.1 per cent. and 31.6 per cent., but that when the total for the two hours is contrasted, the retardation is less, and in two cases practically the same amount was excreted. It will be noted that in Case 3, the figures for the retardation are extreme, and much higher than the average. If this case is eliminated, the slowing taking place in the first hour and the increased excretion in the second hour, which brings the totals nearer for the two positions, is more

TABLE SHOWING PHTHALEIN EXCRETION IN LORDOTIC ALBUMINURIA CASES AND CONTROLS*

Case	Position				Difference While in Lordotic Position		Remarks
	Normal		Lordotic				
	First Hour	Second Hour	First Hour	Second Hour	First Hour	Second Hour	
1	62.8	67.8	42.8	52.8	—20.0	—15.0	Appeared three minutes earlier in normal position.
2	48.8	58.8	39.0	59.0	— 9.8	+ 0.2	
3	66.6	86.6	35.0	40.0	—31.6	—46.6	
4	48.3	63.3	41.2	51.2	— 7.1	—12.1	
5	55.0	70.0	30.0	70.0	—25.0	0.0	Appeared three minutes earlier in normal position.
6	60.0	70.0	45.0	60.0	—15.0	—10.0	Appeared two minutes earlier in normal position.
7	55.0	67.0	40.0	60.0	—15.0	— 7.0	
Average	56.6	69.0	39.0	56.1	—17.6	—12.9	
Control 1...	55.0	70.0	56.5	71.5	+ 1.5	+ 1.5	
Control 2...	71.3	83.8	76.8	81.8	+ 5.5	— 2.0	First hour, lordotic represents seventy minutes, instead of one hour.

* Figures are in percentages.

striking. Thus, eliminating this case, an average of 15.6 per cent. less excretion takes place in the first hour, and a total decrease of 7.3 per cent. in two hours. A simple way of stating the finding is that cases of orthostatic albuminuria in bed excreted 56.6 per cent. of phthalein in the first hour and 12.4 per cent. in the second. In accentuated lordosis, a 39 per cent. output took place in the first hour and 17.1 per cent. in the second.

The normal control cases showed no change in elimination between the normal and lordotic positions.

If the above findings may be regarded as lending support to any of the various theories of pathogenesis, it would probably be that one which assumes a mechanical interference with renal circulation as the chief causative factor in the production of an orthostatic albuminuria. Jehle, who has been the most ardent advocate of this theory, believes that the accentuation of the normal forward curve of the lumbar spine so commonly seen in individuals with orthostatic albuminuria, produces an interference with the renal circulation either by compressing or stretching the renal vessels, or otherwise causing a congestion of the kidneys. He does not deny that vasomotor influences may affect the degree of albuminuria, but believes the mechanical factor to be the main feature, without which there is no orthostatic albuminuria. In support of his contention, he submits evidence to show that effacing the lordosis by postural methods causes a disappearance of the albuminuria, and that, furthermore, it is possible to produce an albuminuria in normal individuals by pressure directed through the abdominal walls against the great vessels, or, in animals, by directly constricting or compressing the renal vessels for a short time. Such methods would produce a retardation of the renal circulation, and would probably delay the excretion of a drug such as phenolsulphonephthalein.

SUMMARY

Renal function, as measured by the phenolsulphonephthalein test, in children with marked degrees of orthostatic albuminuria, is normal when the patients are at rest in bed. When these patients are placed in a position of accentuated lordosis, producing a marked albuminuria, the total output of phthalein in two hours is reduced—in the above seven cases, on an average 12.9 per cent. The most marked feature, however, is the retardation which takes place in the output during the first hour—the average of our cases being 17.6 per cent. less in the lordotic position. Normal children do not show this retardation and decreased elimination with the change of posture. If this retardation may be brought into relation with any of the theoretical ideas of the

pathogenesis of orthostatic albuminuria, it would probably be that which associates the condition with a decreased vascular supply to the kidney as the result of posture.

Metropolitan Building.

REFERENCES

Jehle, L.: Ergebn. d. inn. Med. u. Kinderh., 1913, xii, 808.

Fischl, R., and Popper, E.: Jahrb. f. Kinderh., 1915, lxxxi, 31.

Langstein, L.: Pfaundler and Schlossmann's Handb. f. Kinderh., Ed. 2, iv, 30.

THE INFLUENCE OF CLOTHING ON THE SURFACE TEMPERATURE OF INFANTS*

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INTRODUCTION

The problem of high infant mortality in summer has received much attention, but of the various factors bearing on this problem, such as high outside- and room-temperature, humidity, etc., the factor of clothing has received relatively little attention. As a result of studies on puppies, we pointed out in a previous communication,[1] that the clothing may so seriously interfere with the loss of body heat at moderately high temperatures as to cause the death of the animal. Not that the necessity of scanty clothing for the infant at high temperatures has not been recognized, but it has never been the object of scientific inquiry. For the adult Rubner[2] has furnished very valuable data with regard to the prevention of heat loss by clothing, while for the infant no such data are available.

We therefore set out to furnish some more accurate data showing the influence of clothing on the heat regulatory power of the infant, particularly at high temperatures. For this purpose we determined the surface temperature of various parts of the infant, clothed and unclothed, at moderate and at high room temperatures. When clothed, the readings were taken on the surface of the clothing as well as on the corresponding skin area beneath the clothes. The rectal temperature of the infant and the temperature and humidity of the room were also recorded. With regard to the rate of heat loss by conduction and radiation, the determining factor in the clothed infant is the difference between the surface temperature of the clothes and the temperature of the surrounding air. In the case of the unclothed infant, the difference between the surface temperature of the skin and the temperature of the surrounding air is the determining factor.

METHOD

The rectal temperature and room temperature were determined by means of mercury thermometers, the relative humidity by means of

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* From the Otho S. A. Sprague Memorial Institute Laboratory of The Children's Memorial Hospital.

1. McClure and Sauer: Clothing as a Factor in the Production of Heat Stasis, AM. JOUR. DIS. CHILD., 1915, ix, 490.

2. Rubner: Lehrbuch der Hygiene, 1907, Ed. 8.

wet and dry bulb thermometers. Because of its greater accuracy and adaptability we used the thermo-electric method[3] for the determination of surface temperatures. The apparatus consisted of a 300 ohm D'Arsonval ballistic galvanometer of the type built by Wm. Gaertner & Co. Copper wires connect the posts with the thermo-elements. These thermo-elements, two in number, were made by twisting together fine copper and constantan wires. With both of the thermo-elements at the same temperature there is no swing of the galvanometer. When one is kept at a constant temperature and the other exposed to a different temperature, an electric current is generated which causes a deflection of the mirror of the galvanometer. In our

Fig. 1.—Applicator supporting thermo-element A.

work one of the thermo-elements was kept at a constant temperature in an ice bath, the other was supported in a suitable applicator by means of which it could be brought into contact with the surface the temperature of which was to be determined. The deflection of the mirror of the galvanometer was measured on a scale graduated in degrees centigrade. By means of a resistance box it was possible to obtain a constant deflection of the mirror from day to day under different room conditions. (Differences in room temperatures introduce

3. We wish to express our thanks to Prof. Millikan of the Department of Physics of the University of Chicago for his suggestions and for the use of the apparatus.

slight changes in the thermo-electromotive forces at the binding posts, etc.) The applicator, as seen in Figure 1, was made of celluloid (20 ×1.2×0.2 cm.) with an oval window at one end (1 cm. in its greatest diameter). The thermo-element used on the skin was suspended in the window, the celluloid forming the frame of the window was made as delicate as practicable in order to eliminate, as far as possible, heat loss from the surface of the skin by conduction through this material. This applicator was found to be especially convenient for taking skin temperature readings beneath the clothes. With the apparatus used it was possible to make readings showing a difference of 0.1 C.

THE EFFECT OF CLOTHING ON THE HEAT LOSS BY CONDUCTION AND RADIATION

First we studied the effect of the ordinary clothing on the heat loss of the infant by conduction and radiation. Three infants were studied: One normal breast-fed infant (wet nurse's baby), and two artificially fed infants which had recovered from minor ailments (Table 1). These infants wore the ordinary hospital clothing consisting usually of a thin cotton gown, double breasted cotton undershirt, cotton hose and diaper (cf. tables). Five minutes after bringing them into the examining room, temperature readings were made on the following parts: middle of forehead, right and left cheeks, the surface of the clothing over the sternum and abdomen, the skin surface of the sternum and abdomen beneath the clothing and the dorsum of each hand. Readings on the abdomen were made in the midline half way between xyphoid and umbilicus. The clothing was then removed and five minutes later readings were made over the skin of the same areas, and in addition on the dorsum of each foot. These readings were made at moderate (about 24.4 C.) and at high room temperatures (about 30.9 C.).

The loss of heat by conduction and radiation is proportional to the difference between the surface temperature of the body and the temperature of the surrounding air. In a clothed individual the temperature of the surface of the clothing must be taken as representing the surface temperature of that particular portion of the body in estimating the heat loss by conduction and radiation. Since the surface temperature of the clothing is lower than that of the bare skin of the same area of the child when unclothed (Tables 1 and 2), it approaches more nearly the temperature of the surrounding air, and the heat loss depending on conduction and radiation is less, therefore, in the clothed than in the unclothed infant. That clothing inhibits heat loss, as is well known, is further emphasized by the fact that the temperature of

TABLE 1.—SURFACE TEMPERATURES OF NORMAL—

No.	Name	Age in Mos.	Weight (Lbs.)	Diagnosis	Room		Rect. Temp.	Foreh.	Cheek	
					Temp.	Humid.			R	L
					C	%	C	C	C	C
1	Harriet C. ...	6	15¼	Breast fed.....	24.4	30	37.7	33.2	30.5	29.8
					25.	..	....	32.9	31.8	30.7
2	Harriet C. ...	6	15¼	Breast fed.....	24.4	26	37.7	32.5	30.7	30.5
					24.4	..	....	33.3	32.	31.6
3	Harriet C. ...	6	15¼	Breast fed.....	24.4	27	37.7	31.3	29.3	29.6
					25.	..	....	31.9	31.3	29.6
4	Albert D.	10	16	Recovered from mild diarrhea	23.3	28	37.	32.8	31.1	29.9
					23.3	..	....	32.1	30.6	30.7
5	Albert D.	10	16	Recovered from mild diarrhea	25.5	26	37.1	31.8	30.8	30.
					25.5	..	....	32.	30.7	30.1
6	Albert D.	10	16	Recovered from mild diarrhea	24.4	27	37.2	31.7	29.7	29.7
					24.4	..	....	32.	30.4	30.3
7	Leonard W.	7	15¾	Colored; convalescent tetany.	23.8	26	36.8	30.8	28.8	29.4
					23.5	..	....	31.4	29.4	29.4

TABLE 2.—SURFACE TEMPERATURES OF NORMAL—

No.	Name	Age in Mos.	Weight (Lbs.)	Diagnosis	Room		Rect. Temp.	Foreh.	Cheek	
					Temp.	Humid.			R	L
					C	%	C	C	C	C
1	Harriet C. ...	6	15¼	Breast fed.....	31.	32	37.5	33.7	31.6	31.6
					31.	..	....	33.3	32.9	32.7
2	Harriet C. ...	6	15¼	Breast fed.....	31.	21	37.4	33.	30.4	30.6
					31.	..	....	33.	32.4	32.4
3	Albert D.	10	16	Recovered from mild diarrhea	29.7	21	37.2	32.2	32.	30.6
					31.6	..	....	32.4	32.8	32.6
4	Albert D.	10	16	Recovered from mild diarrhea	30.5	35	37.1	34.3	33.5	33.5
					31.	..	....	34.3	33.9	34.3
5	Albert D.	10	16	Recovered from mild diarrhea	31.6	32	37.2	32.5	33.1	33.7
					31.	..	....	34.4	34.2	34.8

—Infants at Moderate Room Temperatures

Chest			Abdomen			Hand		Foot		Remarks
Over Clothes C	Under Clothes C	Bare C	Over Clothes C	Under Clothes C	Bare C	R C	L C	R C	L C	
29.5	35.5	....	30.7	35.2	....	32.9	33.3	....	....	Cotton undershirt (double breast). Cotton gown, hose, diaper.
....	....	33.4	....	....	33.6	32.5	30.9	30.7	30.7	Bare.
31.6	35.3	....	30.7	35.	....	32.	32.2	....	....	As in No. 1, but hose ½ wool.
....	....	34.	....	....	33.8	32.7	31.8	29.5	29.8	Bare.
30.	34.2	....	30.1	35.1	....	31.6	32.2	....	....	As 2.
....	....	32.6	....	....	33.3	32.	29.6	28.5	29.5	Bare.
31.9	34.8	....	31.5	35.2	....	32.	31.8	....	....	Cotton undershirt (single breast). Cotton gown, diaper, ½ wool hose.
....	.. .	33.	....	....	33.1	30.6	30.7	31.5	30.2	Bare.
31.	34.6	....	31.4	35.5	....	32.3	32.3	....	....	Cotton undershirt (double breast). Cotton gown, diaper, hose.
....	.. .	33.6	....	....	33.7	31.7	31.7	31.7	31.3	Bare.
31.3	34.6	....	31.5	35.7	....	32.6	32.3	....	....	As 1.
....	....	33.6	....	....	34.4	32.3	32.3	32.9	32.8	Bare.
27.6	33.1	....	28.4	34.1	. ..	31.8	31.6	....	....	½ wool double breast undershirt. Cotton gown, hose, diaper.
....	....	32.5	....	....	33.1	30.	30.	28.2	27.6	

—Infants at High Room Temperatures

Chest			Abdomen			Hand		Foot		Remarks
Over Clothes C	Under Clothes C	Bare C	Over Clothes C	Under Clothes C	Bare C	R C	L C	R C	L C	
31.8	35.	....	32.7	36.1	....	32.9	32.7	....	....	Cotton undershirt (double breast). Cotton gown, hose, diaper.
....	....	33.3	....	....	33.7	32.5	32.1	31.	31.	Bare.
30.8	35.4	....	30.4	35.8	....	33.8	32.	....	....	½ wool undershirt, hose. Cotton gown, diaper.
....	....	33.6	....	....	34.	33.	32.2	32.2	32.4	Bare.
30.	34.	....	32.	35.4	....	32.4	32.6	....	....	As No. 1.
....	....	34.	....	....	34.6	32.6	31.8	32.6	32.4	Bare.
32 7	35.3	....	32.9	36.8	....	33.3	32.7	....	....	½ wool undershirt, cotton gown, hose, diaper.
....	....	35.3	....	....	35.7	34.5	33.9	34.3	33.3	Bare.
33.3	36.1	....	34.4	36.7	....	34.2	33.5	....	....	Cotton gown, diaper, ½ wool undershirt, hose.
....	....	35.4	....	....	35.8	34.2	34.2	33.3	33.5	Bare.

the skin beneath the clothing is higher than that of the same area when unclothed (see Tables 1, 2, 8).

The accompanying diagram (Fig. 2) has been constructed from Tables 1 and 2. It shows the difference in temperatures between the air and the bare skin of the chest, in the first place, and the air and surface of the clothing over the chest, in the second place. Bars I and II show the average differences in temperature of the chest of the unclothed child and the air, and of the clothing over the chest of the clothed child and the air, respectively. Bars I and II represent these differences at moderate room temperature (24.4 C.); bars III and IV represent simliar differences for the observations made at the high room temperature (30.9 C.). To be more explicit, for Bar I the average surface temperature over the bare chest was 33.2 C., the average room temperature 24.4 C., a difference of 8.8 C. For Bar II the aver-

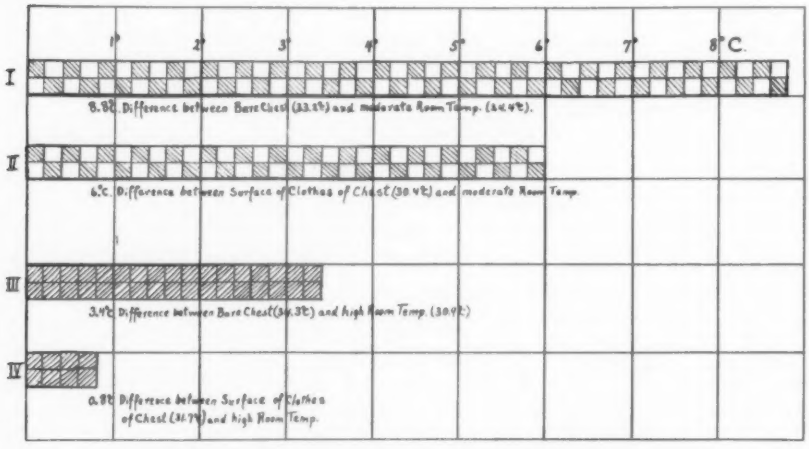

Fig. 2.—Diagram of temperature differences.

age temperature over the clothing on the chest of the clothed infant was 30.4 C., the average room temperature 24.4 C., a difference of 6 C. For bar III the average surface temperature of the bare chest of the bare infant was 34.3 C., the temperature of the room 30 C., a difference of 3.4 C. For bar IV the average surface temperature over the clothes on the chest was 31.7 C., the average room temperature 30.9 C., a difference of 0.8 C.

It is evident that the difference in temperatures of the surface of the body and the room alone does not permit an exact determination of the amount of heat loss by conduction and radiation. At the same time it must be admitted that the heat loss is proportional to this difference. A comparison, therefore, of the differences represented by Bar I and II, and III and IV gives us an indication as to what extent

TABLE 3.—Surface Temperatures of Normal, Unclothed Infants at Moderate Room Temperature

(From Table No. 1, except Cases 8, 9)

No.	Name	Age in Mos.	Weight (Lbs.)	Room T. C	Humid. %	Rect. T. C	Foreh. C	Cheek R C	Cheek L C	Chest C	Abdom. C	Hand R C	Hand L C	Foot R C	Foot L C	Remarks
1	Harriet C.	6	15¼	25.	30	37.7	32.9	31.8	30.7	33.4	33.6	32.5	30.9	30.7	30.7	Normal, breast fed, awake but quiet.
2	Harriet C.	6	15¼	24.4	26	37.7	33.3	32.	31.6	34.	33.8	32.7	31.8	29.5	29.8	Normal, breast fed, awake but quiet.
3	Harriet C.	6	15¼	25.	27	37.7	31.9	31.3	29.6	32.6	33.3	32.	29.6	28.5	29.5	Normal, breast fed, awake but quiet.
4	Albert D.	10	16	23.3	28	37.	32.1	30.6	30.7	33.	33.1	30.6	30.7	31.5	30.2	Recovered from mild diarrhea.
5	Albert D.	10	16	25.5	26	37.1	32.	30.7	33.	33.6	33.7	31.7	31.7	31.7	31.3	Recovered from mild diarrhea.
6	Albert D.	10	16	24.4	27	37.2	32.	30.4	30.3	33.6	34.4	32.3	32.3	32.9	32.8	Recovered from mild diarrhea.
7	Leonard W. ..	7	15¾	23.5	26	36.8	31.4	29.4	29.4	32.5	33.1	30.	30.	28.2	27.6	Colored, convalescent tetany, moderate rickets.
8	Richard M. ...	..	..	22.	33	37.1	31.2	29.2	29.2	33.	33.2	27.6	27.5	28.	28.4	Slight discharge from right ear.
9	Fay M.	11	14	25.5	24	37.4	32.7	31.1	31.1	34.6	34.5	30.9	30.1	31.9	31.1	Convalescent tetany.
	Average.....			24.3	27.4	37.3	32.2	30.7	30.3	33.3	33.6	31.1	30.5	30.3	30.2	

TABLE 4.—Surface Temperature of Atrophic, Unclothed Infants at Moderate Room Temperature

No.	Name	Age in Mos.	Weight (Lbs.)	Room T. C	Humid. %	Rect. T. C	Foreh. C	Cheek R C	Cheek L C	Chest C	Abdom. C	Hand R C	Hand L C	Foot R C	Foot L C	Remarks
1	Harriet J.	6	7½	23.8	28	36.7	34.	32.4	32.9	34.6	33.6	32.6	31.3	31.1	30.2	Atrophic; awake, but quiet.
2	Evelyn G.	2½	4	23.8	25	36.7	34.2	32.4	32.9	35.4	35.8	31.9	31.3	27.1	26.9	Atrophic; awake, but quiet.
3	Vern B.	4	4½	25.	24	37.3	34.1	33.3	33.9	35.1	34.5	32.7	31.3	32.3	32.1	Atrophic; pyelocystitis (only occasional flareups of temp.); awake, but quiet.
4	Vern B.	4	4½	25.5	..	35.4	33.6	33.3	33.9	34.9	34.6	32.9	32.1	32.7	32.4	Atrophic; pyelocystitis (only occasional flareups of temp.); awake, but quiet.
5	Vernet G.	3½	4¾	25.	28	37.2	33.5	32.3	33.1	34.4	35.	30.3	29.9	30.1	29.7	Atrophic; awake, but quiet.
6	Helen P.	4	6¾	23.8	26	36.6	33.6	32.2	32.8	34. —	34.4	32.1	31.2	32.4	32.4	Atrophic; awake, but quiet.
7	Marie A.	3	5¾	23.8	26	35.4	34.	32.8	33.4	34.2	34.	31.5	32.1	32.5	32.8	Atrophic; awake, but quiet.
	Average.....			24.4	26.1	36.5	33.9	32.9	33.3	34.7	34.6	32.	31.3	31.2	30.9	

TABLE 5.—Surface Temperature of Other Infants with Inanition. Bare at Moderate Room Temperature

No.	Name	Age in Mos.	Weight (Lbs.)	Room T. C	Humid. %	Rect. T. C	Foreh. C	Cheek R C	Cheek L C	Chest C	Abdom. C	Hand R C	Hand L C	Foot R C	Foot L C	Remarks
1	Evelyn E.	2½	6¾	23.8	24	36.8	32.4	32.	32.6	33.4	33.8	32.	31.8	32.4	32.4	Pyloric stenosis.
2	Robt. H.	3	6¼	23.3	..	35.4	33.4	32.1	31.7	32.6	33.2	29.1	28.8	28.5	29.5	Pyloric stenosis.
	Average.....			23.6	24	36.1	32.9	32.1	32.2	33.	33.5	30.6	29.9	30.5	31.	

the heat loss by conduction and radiation is influenced by clothing at the respective room temperatures. If we let Bar I equal 100 then Bar II would equal 68.2; likewise, if we let Bar III equal 100, then Bar IV would be equal to only 23.8.

If we accept these figures, with all due reservation, as the basis of calculation for the heat loss by conduction and radiation, it would then become evident that the clothed infant at a moderate room temperature is capable of availing itself of about 68 per cent. of the possible heat loss due to these factors. At the high room temperature it can only avail itself of about 24 per cent. of this possible heat loss, and this in spite of the fact that the surface temperature of the bare chest at the

TABLE 6.—AVERAGES
Bare at Moderate Temperatures

	Normal	Atrophy	Pyloric Stenosis
Room temperature	24.3 C	24.4 C	23.6 C
Room humidity	27.4%	26.1%	24 %
Rectal temperature	37.3	36.5	36.1
Forehead	32.2	33.9	32.9
Right cheek	30.7	32.9	32.1
Left cheek	30.3	33.3	32.2
Chest	33.3	34.7	33
Abdomen	33.6	34.6	33.5
Right hand	31.1	32	30.6
Left hand	30.5	31.3	29.9
Right foot	30.3	31.2	30.5
Left foot	30.2	30.9	31

high room temperature is 1.1 C. higher than at the moderate room temperature. The rectal temperature is about the same at both moderate and high room temperatures. The rise of the skin temperature favors heat loss by conduction and radiation, but this is more than counteracted by the clothing.

The surface temperature of the clothing over the chest averaged about 2.7 C. lower than the surface temperature of the chest of the unclothed infant. This difference was about the same at moderate and at high room temperatures. Judging from these data we would expect the clothed child to cease losing heat from this area by conduction and radiation in a surrounding temperature of about 2.7 C. lower than would the unclothed infant. With the high room temperature the

clothed child approaches rapidly the point where heat loss by conduction and radiation is no longer possible.

Heat loss, due to evaporation from the body surface, has not received attention in this study; and the question might be raised, therefore, whether the inhibition of heat loss by conduction and radiation due to clothing, might not be compensated for by an increase of evaporation. Against this supposition we find that the surface temperature of the skin beneath the clothing is higher than that of the skin of the same area unclothed, the difference averaging over the chest 0.9 C. for the moderate room temperatures and 1.4 C. for the high room temperature. If evaporation was sufficiently increased to compensate for the decreased heat loss due to inhibition of conduction and radiation, we would expect no such increase of surface temperature under the clothing.

TABLE 7.—TEMPERATURE OF CHESTS OF NORMAL INFANTS
(From Tables 1, 2)

No.	A. Moderate Room Temperature				B. High Room Temperature			
	Room Temp. C.	Bare C.	Over Clothes C.	Under Clothes C.	Room Temp. C.	Bare C.	Over Clothes C.	Under Clothes C.
1	25	33.4	29.5	35.5	31	33.3	31.8	35
2	24.4	34	31.6	35.3	31	33.6	30.8	35.4
3	25	32.6	30	34.2	30.5	34	30	34
4	23.3	33	31.9	34.8	31	35.3	32.7	35.3
5	25.5	33.6	31	34.6	31	35.4	33.3	36.1
6	24.4	33.6	31.3	34.6	....	....	....	....
7	23.5	32.5	27.6	33.1	....	....	....	....
Aver.	24.4	33.2	30.4	34.6	30.9	34.3	31.7	35.2

SURFACE TEMPERATURE IN DISEASE

In the course of our work we made surface temperature readings on infants with various types of inanition (atrophy and pyloric stenosis). The children were brought into the examining room, all of the clothing was removed, and after five minutes the surface temperatures were read. We give below the data obtained from the two groups of these cases (Tables 4 and 5). In order to compare these with the findings in the normal infant, we have constructed Table 7. The findings in the cases of atrophy, in which the rectal temperatures were considerably lower than in the normal infants, were rather surprising in that all the surface temperature readings showed a higher

average than those for the corresponding surfaces of the normal infants. This higher surface temperature, causing greater heat loss, would lend support to the findings of Schlossmann,[4] Bahrdt and Edelstein,[5] and Murlin and Hoobler,[6] who have shown by calorimetric methods that the metabolism of the atrophic infant proceeds at a higher level than that of the normal infant. In seeking an explanation for the cause of this high surface temperature, the thinness of the subcutaneous tissue seemed to offer a ready explanation. That this is not the entire explanation, however, is indicated by the surface temperature readings of the pyloric stenosis cases (Table 5), in which emaciation was also very marked. The surface temperature readings approximated very closely those of the normal infant.

We also made temperature readings in a variety of diseases including infections such as pneumonia, cystitis, otitis media, etc., but from the small number of such cases, no conclusions can be drawn at this time.

CONCLUSION

At a room temperature of about 31 C. an infant, clothed in the manner above described and under the conditions of our observations, approaches very closely the point where a heat loss by conduction and radiation is no longer possible. Our experiments with puppies indicate that such a state of affairs may be fraught with danger to the organism.

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4. Schlossmann: Ztschr. f. Kinderh., 1912-13, v, 227.
5. Bahrdt and Edelstein: Festschrift, Dr. Otto Heubner, Berlin, 1913.
6. Murlin and Hoobler: The Energy Metabolism of Ten Hospital Children Between the Ages of Two Months and One Year, Am. Jour. Dis. Child., 1915, ix, 81.

THE AMMONIACAL DIAPER IN INFANTS AND YOUNG CHILDREN *

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I

Several years ago I was asked to see a little girl, 17 months old, suffering from a severe dermatitis of the diaper region. The skin of the buttocks, perineum, genitals, thighs, and lower abdomen was severely inflamed and numerous vesicles and blebs were scattered over this area. The infant appeared perfectly well otherwise. The mother stated that the diaper removed that morning had a very strong odor, "just like ammonia." She had saved the diaper in a closed receptacle. On unfolding the wet diaper, a very strong penetrating odor of ammonia was noticed. So strong was this gas that it irritated my eyes very disagreeably during the examination. It seemed clear to me that the skin lesions were induced by the ammonia in the urine, since ammonia is a powerful rubifacient and vesicant.

This case served to interest me in the study of ammonia in the urine and the resulting lesions, and for many years facts in regard to this condition were collected and form the basis for this study. The term *ammoniuria* was used to designate the presence of free ammonia in the urine, as well as the skin lesions produced by this irritant.

CLINICAL PHENOMENA

The ammoniacal diaper is frequently encountered by the practitioner. Usually, the odor of ammonia is faint, especially in young infants. Only occasionally does the concentration become sufficiently intense to produce the characteristic irritation of the skin. The severest grade occurs in infants between 1 and 2 years of age.

The lesions consist of inflamed areas on the inner side of the thighs, the genitals, the buttocks and lower abdomen. Sometimes the whole diaper region shows a diffuse redness.

More characteristic is the appearance of vesicles and blebs. There may be only one at the end of the prepuce; often this is associated with others on the prominent parts of the genital region. It is the parts which come in direct contact with the diaper which show the blisters. Rarely the whole diaper region may show numerous confluent blisters. No vesication may be observed, but after the intense

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congestion, sometimes the whole epidermis becomes hard, cracked, and the skin seems covered with thin parchment. The dead skin separates in a flaky desquamation.

A very important lesion to which no attention has been given by pediatrists, is the formation of a blister at the meatus. This occurs only in boys who have been circumcised. The prepuce protects the meatus in the uncircumcised. The blister ruptures and a superficial ulceration remains. This causes distress in urinating and may be the source of hemorrhage. The ulcer is exceedingly troublesome at times. It becomes covered by a thin crust, which may partially or entirely occlude the orifice of the urethra, so that urination is painful and difficult. The passage of the urine removes the crust and the ulcer is irritated again by the urine. Healing, therefore, may take several weeks.

The skin lesions nearly always appear in the morning when the infant has lain for a long period during the night. Infants who do not wet the diaper are not subject to this trouble. In fact, enuresis and ammoniuria go together. The assumption that the enuresis depended on the ammonia in the urine was entertained for awhile; the reverse is probably true.

The ammoniacal diaper is a frequent precursor of pyelocystitis in female infants. The irritated genital region becomes more susceptible to infections. On account of the protection afforded by the labia, the urethral orifice in the female is not subject to the blistering.

The association of the ammoniacal diaper and the exudative diathesis is frequent. The irritating urine initiates patches of eczema in the diaper region, which are exceedingly intractable unless the urinary condition is relieved.

The onset is usually abrupt and the course is very irregular and indefinite. The irritating urine may be present for one day only; then, it may last for weeks.

ILLUSTRATIVE CASES

CASE 1.—C. C., girl, aged 8 months; fed on dextrinized flour and milk; has had a tendency to eczema on the face. Lately she has been very irritable and urinates frequently. She is rather constipated, but the nutrition is very good. The mother noticed that the diaper every morning had a strong odor like ammonia. There is considerable irritation of the buttocks, but no blistering.

CASE 2.—B. C., boy, aged 4 years, has nocturnal enuresis. The mother has observed a very strong odor of ammonia. This morning she detected blood stains on the sheets, which alarmed her. Examination revealed the glans penis congested, and an ulcer covered by a crust at the meatus.

CASE 3.—R. D., boy, 1 year old, has been artificially fed since birth. His nutrition is very good at present. His diet consists of whole cows' milk, cereals, bread, custard, orange juice, apple sauce and prune juice. He is usually constipated. He passes much urine through the night; the bedding is soaked in the

morning. Considerable irritation of the genital region has been present for the last two days. This morning the whole prepuce was swollen and edematous, and a large blister is present on the dorsum. Another vesicle is found on the inner side of the thigh. The bedding has a very strong odor of ammonia.

As the ulcer would not heal, after ten days the family had the boy circumcised. After the operation the ammoniuria continued. The head of the penis and wound edges became irritated, swollen and a large blister formed on the glans penis. He had difficulty in urinating and a crust formed at the meatus. The urine continued to give this strong ammoniacal odor for several weeks longer, so that the little boy became known as the "ammonia factory." Treatment had no effect.

CASE 4.—C. A., boy aged 23 months; on a general diet; passed about half a teaspoonful of blood after urinating. The glans penis showed ulceration and a crust covered the meatus, occluding the opening. The mother said the urine was very strong.

CASE 5.—J. P., 2 years old, boy, has always been well. Was breast fed to 11 months. Was seen first in February, 1915. Because he has been housed too much, he looked pale and appetite was poor. He was on a general diet. Was suffering from incontinence of the urine. Clothes wet all the time. He was restless at night. During the last week mother noticed severe irritation of the genital region. One blister was found on the scrotum and the urinary meatus showed an ulceration. The mother observed an ammoniacal odor of the bed clothes. Bowels rather constipated.

CASE 6.—S. L., boy, aged 2 years. Was operated on for adenoid vegetations one week ago. The mother noticed a remarkable penetrating odor of ammonia in the diaper, which alarmed her very much. Very slight genital irritation.

The boy was on mixed diet. Has had recurrent attacks of intestinal indigestion. The mother noticed a similar odor one year before when she changed her washwoman. She was directed to supervise the rinsing of the diapers and the odor from the diapers disappeared.

ETIOLOGY

Ammonia in the urine of infants and young children has been observed by pediatrists. The ammoniacal diaper has become a well-known term, following the suggestion of Southworth. In St. Louis this condition is very frequent. While we pass over the cases in which a slight ammoniacal odor of the bedding is noticed, in the last five years, I have collected brief notes of seventy-eight cases, in all of which the patients showed some skin lesions, and the ammonia was sufficiently intense to warrant more than a passing notice.

Of these seventy-eight cases, fifty-four, or about three-fourths, occurred in children over 10 months of age. The worst cases are observed, not in infants, but in young children, from 1 to 4 years of age. By far the greater number of cases occur in the winter months.

The severest lesions in my series have occurred in boys. As already mentioned, pyelocystitis, not infrequently, follows the occurrence of severe ammoniacal condition in girls. It seems to me this predisposing cause to pyuria should be given more attention.

While several infants were fed partially on the breast, the disorder seems to be almost limited to artificially-fed infants. The severest

grades arise at the time when the infant is beginning to take solid food. Mixtures containing cow's milk and cereal decoctions are especially prone to result in the ammoniacal diaper. Taking large quantities of bread was etiologically related in a few cases. Egg also seemed to be a disturbing factor.

Several careful tests were made to determine the effect of orange juice. In three infants the mothers insisted that the ammoniacal odor was stronger when orange juice was given. Repeated tests in one little girl showed that the diaper was ammoniacal when orange juice was given, and had little odor when the juice was omitted.

High percentages of fat in the milk seemed provocative of this disorder in young infants. The removal of the fat from the food had very little effect on the ammonia in older infants.

Buttermilk, casein-buttermilk, whole milk, and condensed milk stood etiologically related in some cases.

The majority of the infants were constipated; often the stools were reported normal.

The administration of alkalies often aggravated the condition; no relief was observed from their use.

The disorder usually came on suddenly and disappeared as mysteriously. Sometimes it lasted one or two days; at other times, several weeks. Recurrent attacks were frequently observed.

The only disorder apparently connected with this phenomenon was the exudative diathesis, eczema, asthma, etc.

II

For several years I have been trying to ascertain the cause of this trouble. After satisfying myself that this emanation from the bedding was really ammonia (by holding a splinter dipped in hydrochloric acid over the diapers), the further origin of this gas was pursued. That the ammonia was not due to bacterial activity was shown by the fact that the odor was perceptible in a very short time after the urine was passed. Then the odor was different, in that the offensive odor of decomposed urine was absent.

Since Keller discovered that infants with digestive disturbances have a great increase in the ammonia content of the urine, it was at first assumed that the ammoniuria really was a symptom of an acute indigestion, although the clinical symptoms by no means corroborated this view. At any rate, following out the theory of an enterogenic acidosis, the cases were treated by the restriction of fats and the administration of alkalies; but this therapy was so disappointing that it was evident that our theory was erroneous.

We (Dr. Coffin, Dr. Koessel and myself) made numerous tests to determine the concentration of the ammonia in the urine. We availed ourselves of the very simple method described by Rosenbloom[1] of determining the ammonia nitrogen in the urine. Dr. Koessel, at first, made a series of tests in the Bethesda Foundling Home. His results are given in Table 1. The figures give the cubic centimeters of decinormal sodium hydroxid used in titrating 10 c.c. of urine after the addition of neutral potassium oxalate (first column) and after the addition of formic aldehyd (second column). By multiplying the figures in the last column by 0.0014, the quantity of ammonia nitrogen in 10 c.c. of urine may be obtained.

TABLE 1.—ESTIMATE OF AMMONIA NITROGEN IN 10 C.C. URINE OF BETHESDA BABIES

Case	First Titration *	Second Titration	Remarks
1	3.5	2.5	
2	1.0	1.0	
3	1.0	0.8	
4	3.1	4.9	Intoxication
5	1.3	1.3	
6	1.6	1.2	
7	1.5	2.6	Diarrhea
8	2.0	1.1	
9	2.4	1.6	
10	3.8	3.1	Constipation
11	1.3	0.6	
12	0.8	0.3	
13	1.7	1.2	
14	2.2	0.9	
15	3.4	1.6	
16	2.4	8.4	Green Stools
17	1.6	0.8	
18	3.8	7.0	Intoxication
19	3.5	2.1	
20	3.7	3.8	Intoxication
21	6.3	6.0	Diarrhea
22	2.2	1.1	

* The figures give cubic centimeters decinormal sodium hydroxid. Infants less than 1 year of age.

It will be seen that the concentration of ammonia nitrogen in infants is not high, as a rule, yet infants with digestive disturbances show a high percentage of ammonia nitrogen in the urine.

Then Dr. Coffin made a series of tests on older children who presented themselves at the clinic or office. The results are shown in Table 2.

1. Rosenbloom, J.: Clinical Methods for Estimation of Total Nitrogen and Ammonia Nitrogen in Urine, Jour. Am. Med. Assn., 1913, lxi, 87.

The ammonia nitrogen in children is relatively higher than in infants. Occasionally a surprisingly high figure may be obtained without apparent cause.

We have examined the urines of a few children who have shown symptoms of ammoniuria. Obviously, the urine which caused the irritation could not be examined. These figures are given in Table No. 3.

TABLE 2.—AMMONIA NITROGEN IN URINES OF OLDER CHILDREN

Initials	Age Years	First Titration	Second Titration	Diagnosis
F. S.	1	3.7	7.2	Marasmus
A. W.	3	2.3	3.9	Enuresis
W. W.	13	6.3	7.4	Bronchitis
F. P.	4	2.3	3.9	Vaginitis
R. S.	1	3.6	0.3	Partial starvation
F. N.	6	5.1	4.0	Neuritis
A. L.	10	11.5	9.5	Acute indigestion
M. E.	.....	5.3	4.3	
J. M.	¾	3.1	1.9	Malnutrition
L. K.	11	4.5	6.9	Enuresis
M. B.	5	2.0	2.2	Hemorrhagic nephritis
E. G.	3	5.6	3.3	Pyelitis
V. W.	3	3.6	3.0	Acute nephritis
L. B.	2	3.5	2.6	Healthy
S. B.	6	4.1	4.2	Enuresis
S. S.	3	5.3	8.9	Asthma
H. F.	6	4.2	5.4	
M. S.	4	4.6	4.8	Enuresis
S. S.	6	6.1	4.6	Enuresis
S. H.	.....	1.5	1.2	
E. S.	.....	4.0	4.0	Enuresis
E. W.	4	3.9	3.7	Eczema
F. H.	1	4.0	4.2	Mucous colitis
R. S.	2	7.0	5.8	Indigestion
D. B.	5	4.6	2.7	
W. D.	3	5.4	6.9	Anorexia
B. M.	2	1.5	2.8	Cystitis
J. P.	2	4.5	3.1	
A. P.	2	8.0	3.6	

As will be seen, the ammonia content is rather high, but not higher than in some other children who did not present the symptoms.

As the concentration of the total solids might account for the variation in the figures, we compared the specific gravity with the ammonia content. Table No. 4. In general, a high ammonia content is found in the urine with high specific gravity.

We made a few tests to ascertain what effect rest and activity have in the ammonia content. Table No. 5.

TABLE 3.—Showing the Acidity and Ammonia in the Urine of Children Who Had the Symptoms of Ammoniuria

Initials	Age Years	First Titration	Second Titration
H. B.	2	6.9	5.8
E. G.	3	5.6	3.3
L. B.	2	3.5	2.6
E. D.	3	4.6	5.1
A. J.	2	5.4	4.8
R. S.	1	7.0	5.8
J. P.	2	4.5	3.1
S. L.	2	2.0	2.2
E. J.	2	3.3	6.0
S. L.	2	2.0	2.4

TABLE 4.—Showing Quantity of Ammonia in the Urine of Children, as Compared with the Specific Gravity

Initials	Age Years	First Titration	Second Titration	Specific Gravity
A. L.	9	11.5	9.5	1.025
H. S.	8	2.2	2.4	1.010
P. A.	5	6.2	4.8	1.014
P. A.	5	1.7	2.4	1.022
S. H.	3	1.5	1.2	1.020
E. W.	4	4.6	3.9	1.020
R. S.	2	4.0	2.7	1.014
D. D.	6	4.4	4.0	1.015
P. G.	2	3.2	4.9	1.012
H. S.	8	1.4	1.7	1.008

TABLE 5.—Showing the Difference in the Acidity and Ammonia Content in the Urine Passed After a Night's Rest, and Later During the Day

Initials	Age Years	First Titration	Second Titration	Diagnosis
P. A. (rest)	4	6.3	7.4	Nephritis
(active)		4.0	5.3	
A. S. (rest)	9	2.8	3.0	Orthostatic albuminuria
(active)		3.5	2.8	
A. J. (rest)	1	6.3	7.2	Ammoniuria
(active)		3.0	2.5	
A. R. (rest)	9	4.0	4.5	Cystitis
(active)		3.1	4.7	

This was suggested by the fact that the morning diaper is the one usually showing the strongest odor of ammonia. It is curious that all but one of the patients so examined showed the ammonia nitrogen higher in the morning after the night's rest. As a rule children who have the symptoms of ammoniacal urine have a high content of ammonia nitrogen in the morning urine.

But this by no means solves the difficulty. This ammonia is combined with acids, but the clinical evidence proves that the ammonia, which causes mischief, is *free* ammonia. We thought at first that the young child, under certain conditions, passes free ammonia, and thus arose the term ammoniuria. While it is clear enough that free ammonia gas was arising from the diaper, we never could obtain a specimen of urine in a vessel or bottle which gave off free ammonia, except in traces. We could not prove, in a single instance, that a child ever passes ammonia in its free state.

It is only recently that I succeeded in demonstrating the origin of the free ammonia in two cases. It is not necessary to give the details. The immediate cause of the ammoniacal diaper is the presence of an alkali in the diaper or bedding.

This fact was tested at the bedside and at the laboratory. When the diaper, which has been washed in a strong alkaline soap, is not thoroughly rinsed in clear water, sufficient alkalinity remains in the cloth to decompose the ammonia in the urine. This is the origin of the "common" saying that strong soap or lye in the diaper blisters the baby. It is not the alkali or soap on the skin, but the ammonia produced, which causes the skin irritation. An alkaline stool mixed with urine acts the same way, and we have often attributed an intertrigo to irritating feces, when it was really caused by ammonia.

It is clear that the neutral or nearly neutral urine would be the most dangerous, since a highly acid urine would neutralize all the alkali before the ammonium salts would be decomposed.

This fact also explains the failure of dieting and the administration of alkalies. It would explain the action of orange juice, since this makes the urine less acid. Its more frequent occurrence in older children, its occurrence in the winter months, and the irregular course, are easily explained when we adopt this theory. One mother told me that her baby invariably passed ammonia after a certain woman washed the diapers. Another mother had no more trouble when she was told to oversee the washing of the nursery bedding.

In St. Louis, on account of the high content of lime in the water, it is possible that this alkaline earth may be a possible cause in certain babies, since in drying the bedding considerable lime would remain.

CONCLUSIONS

Free ammonia in the diapers of young children is a frequent condition.

If much ammonia is present, severe irritation and vesication of the diaper region may occur.

The ammonia is derived from the ammonium compounds in the urine, and is liberated by an alkali present in the diaper—soap, lye, lime, or stool.

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CLINICAL DEPARTMENT

ACUTE MYELITIS FOLLOWING VARICELLA

REPORT OF A CASE *

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It is believed that almost any of the infectious diseases may attack the central nervous system to produce encephalitis or myelitis. The following is a list of those mentioned: Typhoid fever, typhus fever, erysipelas, malaria, variola, measles, acute rheumatism, severe puerperal infections, scarlatina, influenza, pneumonia, pertussis, dysentery, cholera, gonorrhea, streptococcus and staphylococcus infections. Only one author (Councilman) has in recent years mentioned varicella as a possible cause. The case that I am now reporting bears such a close relationship to an attack of varicella that I consider it of sufficient interest to record.

History.—The patient, R. T., a white boy, 7 years of age, applied for treatment at the dispensary of the Harriet Lane Home, Aug. 17, 1914, complaining of "stiff knees."

Family History.—Unimportant.

Personal History.—The patient was a full term child, following a normal delivery. He was breast fed for eighteen months, and cut his first tooth at seventh month. There was an abscess behind the left ear at the eighth month, which was not serious, and pertussis at 5 years. These were the only two illnesses that he had had, and with these two exceptions, the patient has always been a healthy child.

Present Illness.—Ten weeks before, June 1, 1915, the patient had an attack of varicella, just after his older brother had recovered from a similar one. He had the usual papulovesicular eruption which was very wide-spread.

Eight weeks before, June 14, 1915, or two weeks after the onset of varicella, the patient's temperature began to rise, and his mother noticed that his legs were drawn up. It was further discovered that he was unable to move either of his legs or his left arm. There was also loss of sensation over these parts, which was more marked over the left leg than over the right. This last condition persisted for two weeks, and sensation gradually returned again. His left arm was held over his head, and he would scream with pain if one attempted to pull it down or to straighten out either of the legs.

A few days after the onset of these symptoms, the child suffered with retention of urine and was catheterized twice. This was followed by incontinence of both urine and feces. This incontinence continued for six or seven weeks. On the first application, or eight weeks after the attack, it was found that under ordinary conditions the sphincter control was good; however, the urine could not be long retained.

* Submitted for publication Oct. 8, 1915.

The patient was admitted to the Harriet Lane Home, August 19, 1914.

Physical Examination.—The child was undernourished but did not look sick. His head, heart, lungs, abdomen and genitalia were apparently normal.

The knees were flexed and the legs could not be entirely straightened. The muscles were soft and perhaps slightly atrophied. Both knee-kicks were hyperactive, the right more so than the left. There was an ankle clonus on both sides, which was better sustained on the right. The muscular strength was good and equal in the arms. The legs were very weak, though they could be moved fairly well voluntarily. Sensations of touch, pain, and temperature were apparently normal. There was some hyperesthesia of both legs.

Of laboratory findings, the von Pirquet skin test was questionably positive, the spinal fluid normal, and the Wassermann negative in both spinal fluid and blood. The eyegrounds were found to be normal.

Sept. 4, 1914, the patient was discharged from the hospital, improved.

Oct. 2, 1914, he returned to the dispensary. He was able to walk slowly and with some difficulty, with both his knees somewhat flexed. The steps were short and the feet were dragged along the floor. He was still suffering with urinary incontinence if there was any delay in reaching the toilet. There was, however, no incontinence of feces.

On physical examination the legs looked thin, as though some atrophy had taken place. The tendon reflexes were still greatly increased. Babinski's test was positive on both sides. There was an ankle clonus present on the right.

Aug. 5, 1915, one year after the first admission, the patient appeared greatly improved and was able to play freely. There was still some definite spasticity noticeable in the lower extremities, best brought out after running or walking rapidly. He was able to extend his legs normally. The tendon reflexes were hyperactive as before, and the Babinski was positive on both sides. There was no ankle clonus, and no evidence of urinary incontinence except at night, a condition which had existed prior to his illness.

The occurrence of these symptoms indicated unmistakably a definite lesion in the cord. They followed so closely on an infection with varicella that it seems justifiable to consider these two conditions as having a definite relationship, especially as there was an absence of any external condition to which might be assigned an etiologic significance.

PROGRESS IN PEDIATRICS

RESUME OF THE RECENT LITERATURE ON RESPIRATORY DISEASES *

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NEW YORK

EXAMINATION OF THE CHEST

Wood[1] believes that the present method of percussion is of little value, because there is no universal standard. He has found that percussion notes can be determined with fair accuracy on the basis of pitch alone. The difference in pitch between normal resonance and flatness is about two octaves. These notes are easily acquired by a little practice by whistling or humming the note percussed. He also believes that the lungs should be percussed from below upward as the normal lung is more apt to lie below, while disease is more common at the apex.

Bauer[2] has found that cases of typical acute food intoxication all showed hyperresonance and an increase of lung area which could be demonstrated, the anterior border reaching to the sixth or seventh rib, overlapping the heart, the posterior border reaching to the eleventh or twelfth dorsal vertebra. Patients who recovered showed a diminution of the size of the lungs.

Mielke[3] has noted a dulness at the apex of the lungs which is due to the weakness of the muscles and faulty posture, and which disappears on making the patient stand erect.

BACTERIOLOGY

Tunnicliff[4] has found an organism which is apparently the cause of acute rhinitis. It is a delicate, curved, gram-negative, anaerobic bacillus which is found in the early stages of acute rhinitis before it becomes purulent. It somewhat resembles a spirochete but is probably a bacillus. She found it in 79 cases of acute coryza, 8 times in pure culture, but in only 5 out of 63 smears from the normal nose. It produced rhinitis in man and in dog. It was not found in purulent

* Submitted for publication Nov. 6, 1915.

1. Wood, N. K.: Jour. Am. Med. Assn., 1914, lxiii, 1378.
2. Bauer, F.: Monatschr. f. Kinderh., 1914, xii, 510.
3. Mielke: Berl. klin. Wchnschr., 1914, p. 1218.
4. Tunnicliff, Ruth: Jour. Infect. Dis., 1915, xvi, 493.

secretions, nor in scarlet and diphtheria. In chronic nonpurulent rhinitis it was found in 17 of 18 cases. It was twice obtained from the sputum in bronchitis.

In an effort to find the cause of coryza, Kruse[5] made a Berkefeld filtrate of the dilated nasal secretions of persons suffering with coryza. The filtrate was bacteria-free. When this filtrate was applied to the nasal mucosa of healthy persons it caused an acute coryza in seventeen out of fifty-four cases. The incubation was one to four days, usually two or three days. In some cases there was also bronchitis. He concludes that the cause of at least one form of coryza and coughs belongs to the ultramicroscopic group of organisms.

Cecil[6] has found that the *Streptococcus viridans* can be isolated from practically all mouths and throats. It is the most common organism found in the infected tonsil. It was also found in all cases of pyorrhea studied, in five out of twelve cases of coryza, and in three cases of sinus disease. In bronchitis eight out of fourteen cases showed a predominance of this organism. All of the viridans infections are mild, but tend to become chronic, are followed by little or no immunity, and hence tend to recur. Vaccine therapy is valuable to prevent recurrence.

Brückner, Gaehtgens and Vogt[7] believe that the influenza bacillus is not a harmless saprophyte, but that it is very frequently the cause of respiratory infections in children. The influenza infections tend to relapse, and often become chronic.

Holt[8] believes that the influenza bacillus is definitely pathogenic in many respiratory infections. As a rule it has not a high degree of virulence, but in exceptional instances this may be the case, and it may cause a general infection. In the Babies' Hospital, the *Bacillus influenzae* was found in 30 to 40 per cent. of the sputum cultures made in the last five years. Pneumococcus was found in 62 to 87 per cent., streptococcus, 29 to 46 per cent., and staphylococcus, 76 to 90 per cent. The belief of many pathologists that the influenza bacillus is not pathogenic is refuted by finding it in pure cultures in meningitis. In influenza infections the wide fluctuation of temperature is the most characteristic feature. This is seen in pneumonia, otitis, cases with few catarrhal symptoms, as a complication of tuberculosis, and in cases resembling whooping-cough. Diagnosis between spasmodic cough in influenza and whooping cough can at times be made only with great difficulty. The blood count differs in the two

5. Kruse, W.: München. med. Wchnschr., 1914, lxi, 1547.
6. Cecil, R.: Laryngoscope, 1915, xxv, 97.
7. Brückner, Gaehtgens and Vogt: Jahrb. f. Kinderh., 1912, lxxvi, 417.
8. Holt, L. E.: Arch. Pediat., 1914, xxi, 724.

conditions, influenza giving a polymorphonuclear percentage of 60 to 70. Prognosis in uncomplicated influenza is good. When it complicates pneumonia, the course is likely to be greatly protracted. Relapses are common in all influenza infections. These cases should be carefully isolated as they are very contagious. They improve rapidly if they can be moved to a warm climate or with the advent of warm weather.

Chappell and Brown[9] call attention to the great frequency and extreme seriousness of respiratory infections in infants' wards. These infections are more serious than common contagious diseases and more difficult to control. The pneumococcus and streptococcus infections cause most of the trouble. Staphylococcus and influenza are also frequently found. They attempt to prevent respiratory infections by increasing the child's resistance by attention to its nutrition, by careful nursing and by postnasal douching. A special syringe was used which throws the stream forward through the nasal cavity. Argyrol, boric acid and peroxid solutions were used. The ward in which this douching was carried out twice a week for each patient, showed a distinctly smaller number of infections than the control ward in which there was the same class of cases. [Further experience with this method at the Babies Hospital has not seemed to bear out the earlier results.]

STRIDOR

Rach[10] enumerates as the varieties of stridor:

1. Nasal stridor, which is snuffling, seen in coryza and adenoids, and which disappears when the nostrils are occluded.

2. Pharyngeal stridor, which is snoring, as seen in greatly enlarged tonsils, and scarlet fever, diphtheria, tonsillar or retropharyngeal abscess.

3. Laryngeal stridor, which is crowing, and is seen in pertussis, laryngospasm in tetany, diphtheria of the larynx, pseudocroup, measles, and local laryngeal conditions, i. e., tuberculosis, tumors, edema, or stenosis after intubation.

4. Endothoracic stridor with Roentgen-ray findings:

a. Thymic stridor, which is congenital or begins in the first month, is inspiratory, constant, and rattling, bleating or clucking. There is dulness over or alongside the sternum. The Roentgen ray shows asymmetrical widening of the middle shadow, usually to the right, but occasionally to the left. The lateral view shows the shadow in the usual clear retrosternal space. Thymic hypertrophy may exist without stridor, but when it co-exists it seems to have a causal relation.

9. Chappell, W. F., and Brown, A.: Am. Jour. Dis. Child., 1914, vii, 380.
10. Rach, E.: Ztschr. f. Kinderh., 1914, xi, 1.

Congenital stridor, due to abnormal softening of the laryngeal cartilages, gives the same type as the thymic stridor. [This type (Thomsen's) might better be classified under laryngeal stridor.]

b. Stridor from enlarged bronchial lymph nodes. This is expiratory, regular, loud, and moist, the inspiration being almost noiseless. There is cough, which is shrill, with overtones, and there are apt to be attacks of suffocative dyspnea. V. Pirquet test is positive. There is occasional edema of the face, exophthalmus and dilated veins of the face and chest. A dulness over the sternum or between the scapula and bronchi, bronchophony from the first to fourth dorsal vertebrae. (In this respect see the view of Stoll (AM. JOUR. DIS. CHILD., December, 1912) who believes that whispered bronchophony is not significant unless heard below the fourth dorsal vertebra.) The Roentgen-ray shadows are discrete, rounded, sharply outlined beside the tracheal bifurcation.

c. Stridor from an abscess from carious vertebrae. This may be inspiratory or expiratory; cough and cry are dull and weak. V. Pirquet is positive; the Roentgen ray shows diseased vertebra and there are other symptoms of spondylitis.

d. Asthmatic stridor, in which there is prolonged expiration with piping or rattling sounds, the thorax becoming fixed in the inspiratory condition. There is fever and apathy and great dyspnea. V. Pirquet is negative (v.i. Rongel), the Roentgen ray shows the diaphragm to be low and almost immobile. The hilus shadows may be thickened.

e. Stridor from tracheal displacement, due to fibrosis, fluid, tumors, etc. This gives no characteristic symptom, but can be recognized with the Roentgen ray.

BRONCHITIS AND ASTHMA

Kerley[11] believes that recurrent bronchitis in children is due to faulty metabolism. It occurs in children who have a history of difficult feeding, many of whom are subject to eczema, recurrent vomiting, migraine, etc. His experience leads him to believe that fat and cane sugar are badly borne by all these children, and he thinks it is the inability of the child to oxidize these high energy foods which gives the predisposition to this group of conditions. His treatment is the complete withdrawal of cane sugar. The child is allowed no cow's milk or only small amounts of skimmed milk. Clothing must be of medium weight; warm baths at bed time followed by vigorous rubbing are useful. The bowels must be carefully regulated. Bicarbonate of soda and salicylates and occasionally arsenic are administered. In

11. Kerley, C. G.: Arch. Pediat., 1914, xxxi, 741.

his hands this treatment gives a very good result in 95 per cent. of the cases.

Rachford[12] believes that migraine, recurrent vomiting, recurrent sibilant bronchitis, coryza, asthma and urticaria are all closely related to food intoxications and are curable by dietetic treatment. The foods allowed include beef, mutton, fowl and fish, cereals, bread, vegetables (except tomatoes), cooked fruits, skimmed milk and thick soups. The foods forbidden are sweets, butter fats, eggs, raw fruit, salads, shell fish, tea, coffee, pastry, gravies, cod liver oil and alcohol. After the child has been on the above diet for some time one egg a day is allowed. Gradually other forbidden articles are added and the child is watched to see if symptoms recur. The bowels are regulated by magnesium salts. A great deal of fresh air is insisted on, especially at night. Alkalies are given in the form of sodium bicarbonate, or potassium citrate, for a period of from six to eight weeks. During the attack the tincture of belladonna (2 to 4 minims) is given three times a day. This treatment will control the syndrome in almost every instance, and is also useful in preventing recurrent vomiting or migraine.

Florand, Francoise and Flurin[13] believe that the character of chronic bronchitis is not explicable by the kind of infection, nor by disturbances of circulation, but depends on a diminished resistance which amounts to a veritable weakness of the bronchi. They have found three symptoms in the interval between the recurrent attacks. There is hyperesthesia of the mucous membrane to differences of temperature. Cold air excites cough and slight dyspnea. There are variations in the circulation which give congestion and swelling in the mucous membrane. This results in functional disturbances of the nose, larynx or bronchi. There is also hypersusceptibility to the factors which tend to increase secretions.

Hutinel[14] believes that asthma in infants is not so often due to odors, powders, emotions, etc., but more often to catarrhal conditions following coryza, hypertrophied adenoids and tonsils, bronchitis, or bronchial adenopathy. Eighty per cent. of the cases give a family history of asthma, migraine, obesity, urinary or biliary lithiasis. Children subject to asthma often have migraine in later life. The asthma is the result of a nervous reaction due to a hypersensibility of the respiratory nerves, causing a spasm of the bronchial muscles. There is a predisposition in certain subjects which is either hereditary, or comes from a special temperament, in some cases lithemic, in some

12. Rachford, B. K.: Arch. of Pediat., 1914, xxxi, 488.
13. Florand, Francoise and Flurin: Ref. in Ztschr. f. Kinderh., 1914, viii, 476.
14. Hutinel: Rev. gén. de clin. et de thérap., 1914, xxviii, 23.

cases anaphylactic. Tuberculosis may coexist with asthma, in spite of the general belief that this does not occur. He recommends hygienic treatment, cold baths and the administration of iodids.

Rongel[15] quotes the conclusions of Soca after the examination of 700 asthmatics, that asthma is a bulbar neurosis, the result of an hereditary predisposition depending on varying influences. It is often secondary to infectious disease and also to chronic diseases (cardiopathy, anemia, etc.). Even when secondary it needs treatment as an independent condition, as treatment of the primary disease often accomplishes nothing. Asthma is often a symptom of tuberculosis. Soca found that two-thirds of his cases of asthma had tuberculosis; the frequent coexistence of the two conditions makes it seem possible that asthma is the reaction to tuberculosis in some persons. To try out this theory Rongel tested 100 asthmatics under 16 years of age. Fifty per cent. gave positive V. Pirquet reactions. In the remaining 50 per cent. it was not possible to prove any tuberculosis by V. Pirquet or by examination. These figures go far to disprove the old theory that asthma and tuberculosis rarely exist in the same person. The importance of exposure is shown by the following:

Cases with	V. Pirquet	
Tuberculous antecedents	32 +	16 —
Asthmatic antecedents	11 +	29 —
Neither tuberculous nor asthmatic antecedents	7 +	16 —

[These observations of Rongel and Soca make it evident that the asthmatic is at least as susceptible to tuberculosis as other children. It seems as if the existence of a chronic bronchitis would offer a fertile soil for the implantation of the tubercle bacillus.]

Manirloff[16] sensitized animals with serum of an asthmatic patient, and later injected a solution of Charcot-Leyden crystals, producing death with symptoms of anaphylaxis. The controls, sensitized with normal serum, show no symptoms after injection of the solution of Charcot-Leyden crystals or of normal sputum solution. He believes that the Charcot-Leyden crystals are derived from proteins and that asthma is a pure manifestation of anaphylaxis.

Talbot[17] reports six cases of asthma, traceable directly to eggs, all of which gave urticarial reaction to egg albumin rubbed on the unbroken skin or on a V. Pirquet scarification. The wheal appeared in a few minutes and lasted half an hour. If the reaction is repeated too often, asthmatic attacks may be precipitated. He found that some of his cases were sensitized to other proteins, as horse serum, pollen,

15. Rongel, A.: Arch. de méd. des enf., 1913, xvi, 95.
16. Manirloff: Centralbl. f. Bakteriol., 1912, lxiii, 564.
17. Talbot, F.: Boston Med. and Surg. Jour., clxxi, 708.

etc. He believes that either the sensitization or a tendency to it may be transmitted from mother to child, and this has been proved to be possible in animals. Many, if not all, cases may be immunized to egg by giving small amounts of egg albumin in capsules and gradually increasing the dose. It must be started far below the patient's limit of tolerance and gradually increased, about a milligram a day or less. When the egg idiosyncrasy is cured the asthma stops. It is better to immunize such a patient against egg albumin than to allow him to run the risk of accidentally getting egg in some cooked food, and in that way become acutely ill.

Schloss[18] studied with great care a child who was sensitized to several different proteins—egg albumin, oatmeal, almonds, and other like foods. A great many others were tested but the child reacted only to those distinct groups. He was cured by giving ovomucoid in 2 milligram doses in capsules, gradually increased.

Mathews[19] has found that 90 per cent. of 300 cases of asthma were relieved by removing the cause of chronic suppuration in the nose and retention of nasal secretions. Drainage gave relief in proportion as it was possible to make it complete and free. The pus and mucus, whether sterile or infected from the antra of asthmatic patients, injected into guinea-pigs, sensitized them, and a second dose caused anaphylactic shock. Asthma with hay fever comes on first only on exposure to an irritant; later without this contact. Some other substances must have developed the possibility of acting as an antigen. On account of the relief obtained from drainage of diseased nasal conditions, he believes that the sensitizing agent may be the retained and autolyzed secretions of the nose or nasal sinuses.

Ephraim[20] discusses very thoroughly the theories of asthma. It has been the generally accepted view that asthma is a neurosis, but it is dangerous to state positively that any condition is a pure neurosis, for so many diseases formerly so classified have been proved by newer methods of investigation to have a genuine organic basis. The following reasons have been advanced for classifying asthma as a neurosis:

1. Its appearance in attacks; but this is also true of many organic conditions of long standing, which give symptoms only in acute attacks.

2. The sudden onset of attacks; but really the attacks do not come on suddenly as do convulsions, paralysis, and other nervous phenomena. Actually, asthma comes and goes more gradually. Even granting

18. Schloss, O. M.: Am. Jour. Dis. Child., 1912, iv, 341.
19. Mathews, J.: Trans. Assn. Laryngol., Rhinol. and Otol., 1913, xix, 62.
20. Ephraim, A.: Verhandl. d. Verein Deutsch. Laryngol., 1913-14, p. 79.

the sudden onset, it signifies no more than a secondary excitation of the nervous system starting from a diseased organ.

3. The relatively healthy aspect of the asthmatic is supposed to indicate its functional nature. This is not always true, but even if true, it merely indicates that no destructive organic illness exists, and does not exclude an organic condition.

4. The asthmatic shows signs of other nervous conditions. This is not the observation of most clinicians, but when other neuroses exist they are probably the result of the wearing illness.

5. It has been regarded as a diseased condition of the respiratory center itself. Results of all recent research, however, have shown that there is no single respiratory center, but merely scattered muscle centers, not anatomically but only synergically united. The stimulation of no center or tract will produce asthma. A contraction of the bronchi can be caused by stimulation of the vagus or trigeminus but none of the characteristics of true asthma are produced, i. e., the rhonchi, the secretion, and the typical breathing. These do not appear in severe spasm of the bronchi, caused by anaphylactic poisoning, muscarin, etc. Strychnin poisoning, even when it causes death by suffocation, does not resemble asthma in the least. Stimulation of the nasal mucosa produces asthma, but only in asthmatics in whom there is already a predisposition to it. Stimulation of the nasal mucosa in normal persons merely produces sneezing, lacrymation or coughing, even when the stimulation is intense. It is therefore the asthmatic disposition which renders the attacks of asthma possible.

That there is a real organic basis for asthma is shown by the following:

1. Attacks come on in sleep when the nervous system is at rest.
2. Catarrhal processes precede and induce the attack.
3. The effects of nitrites and iodids, substances which have not the least effect on the nervous system.
4. The sputum, which is thick, tenacious and rich in cells.
5. Necropsy findings regularly show changes in the bronchi.
6. Fever occurs fairly often.
7. The occurrence of asthma in localized areas of the lung.

The one thing lacking to complete the picture of an organic disease in the bronchial mucosa is the continuity of the disease which we are accustomed to see in all organic conditions. Where there is a chronic bronchitis we have the tie which joins one attack to another, but where there are apparently healthy intervals it is more difficult to find this tie.

Ephraim has found that when an endobronchial application of epinephrin is made on an asthmatic in an absolutely free interval, the reaction is different from that in a healthy patient. In the asthmatic

there is an expectoration of a sticky, gray sputum, which was not present before, and which does not appear in a normal person. Microscopically this sputum shows small gray, thread-shaped plugs, suspended in a clear mucus, which resemble ciliated bronchial epithelium. These cells do not stain well, but appear necrotic, and show that there must be a chronic desquamative process present in the asthmatic, even when we can discover no symptoms, and this constitutes the asthmatic disposition. The causes which transform this symptomless chronic catarrh to an asthmatic attack are numerous. As internal causes, he recognizes anything which causes an increased congestion of the bronchi, i.e., horizontal position in sleep, overloading the stomach, abnormally deep breathing, as in coughing and laughing. As external causes, anaphylaxis, idiosyncrasy against spices, odors, etc., overloading the blood with carbon dioxid, reflex and psychic nervous factors. The nerve centers are in a state of increased irritability which is a secondary, definite irradiation to the segments corresponding to the bronchial nerves. The psychic element is merely accidental as in all other diseases of an organic nature, as cardiac disease, tuberculosis, etc. In nasal reflex asthma, the irritable membrane may be related to Head's zones of irritability on the skin in visceral diseases.

In discussing Ephraim's paper Killian believes that there is a form of asthma directly due to nasal hyperesthesia, a form which starts regularly from the bronchi, and a mixed form. Hyperesthesia alone is not enough to start the attacks, but there must be a predisposition, and an excitability of the nervous system. Patients can have hay fever from three to five years before asthmatic attacks begin. The hay fever gradually produces the nervous irritability which is necessary to cause asthma.

[It is evident that there are two distinct view points from which the nature of asthma is considered. Certain cases are definitely the result of anaphylaxis to eggs, serum, pollen or other foreign proteins. Other cases occur in patients who have chronic or recurrent bronchitis and there is no evident relation to anaphylaxis. It is possible that the sensitizing protein in the second group of cases is derived from the bacteria present in the air passages, or from changes in the proteins of the secretions in the air passages, which render the individual's own protein toxic. It may well be that the two types of asthma are quite distinct and that bronchial spasm is merely a condition which may be excited by various causes. For a discussion of the relation of asthma and anaphylaxis see Zinsser, H., Infection and Resistance, McMillan, New York, 1914, p. 434.]

Pirie[21] reported twenty-five cases of bronchitis and asthma, treated with autogenous vaccines. The bacteria used were pneumococci, streptococci, staphylococci, and Friedlander's bacilli. Nearly all were mixed

21. Pirie: Brit. Med. Jour., 1913, 1268.

infections. The vaccines were given up to the point at which a mild reaction was produced. Of the chronic bronchitis cases seven were cured, four moderately improved, and four improved slightly. In nine cases of asthma two were cured and five considerably improved.

Borch[22] recommends a proprietary preparation of pituitary gland, which in most cases relieved the attacks and often prevented recurrences in his hands. He noted unpleasant effects in three children between 11 and 13 years, difficult breathing and collapse. Care should be taken in administration of this remedy in children.

BRONCHOTETANY

Lederer[23] describes a new syndrome which appears in the course of tetany. In spasmophilic children, sometimes as the only symptom, sometimes with other signs of this condition, the bronchial muscles become tonically contracted. The smallest branches of the bronchi are affected most and the alveoli supplied by them are shut off from the outside air. The air imprisoned thus is gradually absorbed so that the affected parts of the lung become atelectatic. A condition of edema supervenes (as in the extremities in carpopedal spasm), and the lumina of the alveoli and bronchi become filled with fluid. He observed this condition in 10 per cent. of his cases of tetany.

His clinical picture is as follows: In the course of a definite tetany, following other symptoms, or occasionally preceding them, increasing dyspnea calls attention to the lungs. There is dulness at the bases combined with tympany above, and over the dull areas is heard high-pitched bronchial breathing with râles. There is an irregular temperature. The dyspnea gradually increases, the patient becomes cyanosed and dies of suffocation. The course may be sudden and shortly fatal, or may come and go over a longer period, but all of his patients died. The diagnosis, if tetany is manifest, is not difficult, but when the bronchial spasm appears as the first symptom, it may be puzzling until other manifestations appear. The signs over any one area may come and go in the protracted cases. The Roentgen ray should differentiate this condition from pneumonia, showing scattered areas of atelectaxis. [The reproduction of his roentgenograms are very poor and show very little. It is doubtful whether the differentiation can be easily made from bronchopneumonia by the Roentgen ray.]

At necropsy areas of atelectasis 0.5 to 2 centimeters in diameter are found, sometimes fusing to occupy an entire lobe; there is compensatory emphysema of the rest of the lung. Section shows empty collapsed alveoli, normal bronchi, full blood vessels and no sign of

22. Borch, I.: Therap. d. Gegenw., 1913, liv, 536.
23. Lederer, R.: Ztschr. f. Kinderh., 1913, vii, 1.

inflammatory reaction. The treatment in his hands was entirely unsatisfactory, as the cases were nearly all too rapid. He recommends human milk, phosphorus, cod liver oil, and carbohydrates. Lederer suggests for this condition the name of *"Bronchotetanie."*

Rietschl[24] reports a case of mild tetany with slightly increased electrical irritability and the peroneus phenomenon, which had recurrent attacks of severe dyspnea. Over the right upper lobe there was moderate dulness and prolonged expiration and rhonchi but no bronchial breathing. The case was thought to be bronchitis with a small patch of pneumonia. The attacks of dyspnea were considered to be asthmatic at first, but against this diagnosis were the signs of the right apex. There was no eosinophilia and epinephrin did not relieve the symptoms. Under treatment with calcium, phosphorus, cod liver oil, the patient gradually recovered. He considers this a case of mild bronchotetany on account of the electrical reactions, the local signs of the lungs and the spasmodic dyspnea. Although the differentiation from pneumonia and asthma is very difficult, he suggests that possibly many so-called asthmatic cases may be bronchotetany. He also divides the cases of asthma in infants into two distinct classes: true asthmatic attacks, which are rare, and cases of recurrent bronchitis with asthma. He has never seen asthmatic bronchitis change into true asthma in children.

Wieland[25] reports cases of bronchotetany and concludes that it is a definite clinical picture in certain severe cases of spasmophilia. It can be diagnosed only by the Roentgen ray shadow which causes a blurring of the lung structures resembling that seen in atelectasis. The prognosis is bad. A milder form, however, exists, which may recover.

Breuning[26] demonstrated a specimen from a case of bronchotetany. There had been a history of convulsion and mild catarrhal symptoms without fever. This was followed by sudden attacks of dyspnea in one of which the child died. At necropsy there was a small amount of pneumonia in the right lower lobe. The rest of the lung showed atelectatic areas with compensatory emphysema. Although this was not a clear case, on account of the presence of pneumonia, he considers it to have been a case of bronchotetany.

LOBAR PNEUMONIA

During the last three years the workers at the Rockefeller Institute have reported epoch-making researches on pneumonia (Cole and

24. Rietschl, H.: Monatschr. f. Kinderh., 1913, xii, 261.
25. Wieland, E.: Monatschr. f. Kinderh., 1915, xiii, 205.
26. Breuning: Ref. in Ztschr. f. Kinderh., 1914, viii, 253.

Dochez,[27] Dochez and Gillespie,[28] Cole,[29] Dochez and Avery[30]). They have found that pneumococci can be definitely divided into groups by their immunologic reactions and by their agglutination with immune serums. By these means they recognize four distinct types of organisms.

Type 1 is composed of pneumococci which are responsible for about 38 per cent. of all cases of acute pneumonia. They are moderately virulent, and develop an immune serum which protects mice against any one of the organisms belonging to the type. This serum also agglutinates all of the group.

Type 2 also produces a serum which gives a high grade of passive immunity, and which agglutinates all the pneumococci of the type. This type is more virulent than Type 1.

Type 3 consists of the *Pneumococcus mucosus* (*Streptococcus mucosus*). There are several closely related races of this type of organism which show cross agglutination, but which do not cause the development of an immune serum. This group is the most virulent of all. The organism is characterized by its thick capsule and by the very thick mucus produced in infections which it causes.

Type 4 is a heterogeneous number of organisms each of which apparently differs from every other. Each one develops a serum which protects against itself, but not against any other pneumococcus. There is no cross agglutination.

The occurrence of the different types in cases of pneumonia is as follows:

	Occurrence, Per Cent.	Mortality, Per Cent.
Type 1	38	25
Type 2	30	36
Type 3	11	47
Type 4	21	6

The incidence and mortality of the various types does not seem to vary greatly from year to year. The same types are found in Boston and Philadelphia, and with about the same relative frequency. There has been no evidence of the change of any organism from one group to another, no matter how long it has been cultivated on artificial mediums, nor after passage through many generations of animals.

The pneumococci which are found in healthy mouths are always of Type 4, except in persons who have been in contact with acute cases of pneumonia, when the type of organism with which that case is

27. Cole, R., and Dochez, A. R.: Trans. Am. Assn. Phys., 1913, xxviii, 606.
28. Dochez, A. R., and Gillespie, L. J.: Jour. Am. Med. Assn., 1913, lxi, 727.
29. Cole, R. I.: New York Med. Jour., 1915, ci, 1; Brit. Jour. Dis. Child., 1915, No. 135, p. 80.
30. Dochez, A. R., and Avery: Jour. Exper. Med., 1915, xxi, 114.

infected may be found. After recovery from an infection with one of the first three types, the organism disappears, and Type 4 reappears as a rule. In exceptional cases the original infecting organism may be found after recovery, but the longest time for which this has been observed was ninety days. Such persons are true carriers. Local changes in the lung are not necessary to make an individual susceptible to infection with the first three types, but are apparently necessary to start an infection with Type 4.

These facts have the greatest importance in the prognosis and treatment of pneumonia. By injecting a solution of sputum in salt solution into the peritoneal cavity of a mouse, and after four or five hours washing out the peritoneum with salt solution, and centrifuging, it is possible to get an abundant growth of pneumococci. The type of the organism can be determined by agglutination tests with the immune serum of Type 1 or 2, or by its morphology if it is of Type 3. If it gives no agglutination with the serums, and does not resemble the mucosus type, it belongs to Type 4.

When the type has been determined, much can be said as to the prognosis. As may be seen from the above figures, it is very good in Type 4, less good in Types 1 and 2, and very bad in Type 3.

[It has been a wonderful experience to be able to give a prognosis in several cases of acute pneumonia with the certainty which this work has made possible. In one case in a child, which lasted seventeen days, the organism was determined to belong to Type 4 and a good prognosis given throughout in spite of a severe infection. The prognosis proved to be correct. If it is true that most cases in children belong to Type 4, as seems likely from the findings in scattered cases in the various New York hospitals, it may be one of the factors which has made the prognosis so good in children.]

The treatment with serums is confined entirely to Types 1 and 2, which produce very active serums. Type 4 produces an active serum, but each one protects against the organism from which it was produced and that alone. The reasons for the failure to obtain any results of consequence in the past is very readily seen. If the disease is caused by Type 1 or 2, serum should be administered in large doses—80 c.c. every twelve hours. The effects of the serum may be judged by the effect on the blood cultures. When they are positive before injection, they become negative after it. The most satisfactory results are obtained with Type 1. In this group the mortality of patients treated with serum shows a marked reduction over nonserum cases. The results in Type 2 cases are not so encouraging, but pneumonia is a disease which varies greatly from season to season and it is impossible to draw any final conclusions until many more patients have been treated.

Peabody[31] found that in rabbits with a severe pneumococcus bacteremia the oxygen combining power of the blood falls progressively up to death. Coincidentally there is even a more marked fall of the oxygen content of the blood. This is due to the conversion of hemoglobin into methemoglobin.

Peabody[32] found that in most cases of lobar pneumonia the decrease in the respiratory surfaces is compensated for by the more rapid respiration, and the oxygen content of the blood is within normal limits. In occasional cases the oxygen content of the venous blood is low and the carbon dioxid content is high, due to interference with the respiratory exchange. In fatal cases there is a progressive diminution in the oxygen content and oxygen combining capacity. These cases usually have an intense bacteremia. There is a formation of methemoglobin, so that hemoglobin can no longer take up and give off oxygen easily. This is probably a factor in the immediate cause of death in many cases.

Wollstein and Meltzer[33] have produced an experimental pneumonia by the insufflation of various organisms into the trachea. Virulent streptococci always produced lobular pneumonia. The walls of the bronchi and the connective tissue framework of the lung were markedly infiltrated with leukocytes. This lobular bronchopneumonia occasionally fused to resemble lobar pneumonia, but there was very little fibrin in the exudate and no pleurisy. Pneumococci insufflated into the trachea in the same manner always gave a lobar pneumonia with a large amount of fibrin, no infiltration of the connective tissue framework, and was usually accompanied by a pleurisy.

Kline and Winternitz[34] found that in order to produce pneumonia in rabbits by insufflation it must be forcibly done, and the catheter must be inserted deeply into a bronchus, as insufflation into the trachea alone does not suffice. After administration of alcohol and exposure to cold, or inhalation of irritating gases, pneumonia was easily produced by insufflation of micro-organisms into the trachea alone. After section of both vagi, pneumonia results even without the insufflation of organisms. They think that possibly the predisposing factors act by their influence on the vagus control of the upper air passages.

Weill and Mouriquand[35] have made important studies of pneumonia by means of the Roentgen ray, and have done much to settle the long disputed questions concerning the diagnosis of lobar pneu-

31. Peabody, F.: Jour. Exper. Med., 1913, xviii, 1.
32. Peabody, F.: Jour. Exper. Med., 1913, xviii, 40.
33. Wollstein and Meltzer: Jour. Exper. Med., 1913, xviii, 548.
34. Kline and Winternitz: Jour. Exper. Med., 1915, xxi, 304.
35. Weill and Mouriquand: Lyon méd., 1912, cxix, 1018.

monia in infancy. The earlier writers affirmed the frequency of pneumonia but confused the different forms. A reaction followed and Parrot denied the existence of lobar pneumonia at early ages. This view prevailed throughout the profession with few exceptions until recently (Comby and D'Espine). The reason is that there has been no certain way to differentiate them until the perfection of Roentgenoscopy. Weill and Mouriquand's first report was of 52 cases of lobar pneumonia. Before the Roentgen ray, in twelve years they had records of only 19 cases, of which only 7 gave signs. Since the Roentgen ray they have had 33 cases, of which 31 gave positive shadows. Of these, 13 cases gave shadows before the appearance of physical signs, 18 cases had a shadow and signs at the same time, and two gave no physical signs at any time. The shadow in all the cases in the upper lobes was triangular, the base at the periphery, usually in the axilla, the apex at the root of the lung. In the lower lobes the heart and liver obscure the triangular form.

Weill and Mouriquand[36] believe that they can divide pneumococcus infection into three classes:

1. Pneumococcus infection without localization in the lung, which is the so-called "abortive pneumonia." This form gives all the general symptoms of pneumonia, the sudden onset, the typical temperature curve with a crisis, herpes of the hips (the vesicles containing pneumococci), and often a fibrinemia. There are no signs in the lungs except perhaps a few fugitive râles, and there is no shadow with the Roentgen ray. The duration is usually shorter than other cases but may be prolonged. These cases occur in the first and sometimes in the second year, and the prognosis is very good.

2. Pneumococcus infection with localization in the lung shown only by the Roentgen ray. This corresponds to the so-called "central pneumonia." With a typical shadow there may be no physical signs to the most careful and repeated examinations over the exact site of the shadow. Some of the cases may give signs late in the disease, but some have no signs at any time in its course.

3. Pneumococcus infections with shadow and signs at the same time.

The signs when present are often insignificant. A few fugitive râles and diminished vesicular breathing high in the axilla are often the only positive findings. The examination should be most careful where statistics have shown the localization to be most common, at the right apex, left base, right base, left apex. The physical signs are more frequent in the lower lobes; the cases without signs are

36. Weill and Mouriquand: Paris méd., Dec. 7, 1912, p. 17.

nearly always apical because the upper part of the thorax moves less with respiration and with partly occluded bronchi the signs are less apt to be transmitted. The general symptoms are usually present for three days before the shadow appears, and the appearance of the shadow is sudden and may be as late as the fifth or sixth day. Rarely there is a precocious localization in the lung, suddenly a part of one or more lobes becoming solid. Such a consolidation dominates the prognosis as in adults. The evolution of the shadow usually takes place in a characteristic manner. It begins at the periphery and extends inwards to the hilus of the lung, forming a triangle with its base in the axilla. At times the primary triangle spreads to involve the entire lobe, but in resolution the triangle reappears. The shadow persists two to three weeks after defervescence. Bronchopneumonia gives no shadow even when of the pseudolobar type.

Weill[37] has made further studies on the differentiation between lobar and bronchopneumonia of the pseudolobar type. He has found that there is practically no way of distinguishing these conditions clinically. Bronchopneumonia usually develops more slowly, there is often more general bronchitis, but atypical cases of lobar pneumonia may behave in the same way. He has noted that there is more commonly retraction of the suprasternal and substernal soft parts in bronchopneumonia, but this may occur in lobar pneumonia with a complicating bronchitis. An inverse type of respiration is frequently seen in bronchopneumonia—a type in which there is a pause between inspiration and expiration. There is seen a peculiar cough in about one-quarter of the cases, a short cough with each expiration for twenty to thirty minutes. All these symptoms may be seen in lobar pneumonia, especially when there is a severe bronchitis. Even the temperature curve may have an irregular form with wide excursions in lobar pneumonia.

The Roentgen-ray shadow differentiates the two forms absolutely. Further experience confirms the constant existence of the characteristic shadow in lobar pneumonia and the absence of shadow in bronchopneumonia, even when it involves a whole lobe and appears solid at necropsy. Occasionally a case occurs with all the clinical symptoms and with transient physical signs, but with no shadow. These are probably cases of congestion which do not go on to complete hepatization. Weill believes this, because he has found that the physical signs do not correspond exactly to the area of the shadow, but are more extensive, as if the congestion surrounding the hepatization were capable of giving changes in the percussion and breath sounds. [This

37. Weill, E.: Méd. mod., 1913, xxii, 1.

really makes a fourth type which should be added to those above, i. e., signs without shadow.]

At necropsy it is very difficult to distinguish the pseudolobar from the true lobar. Weill found that by injecting formaldehyd solution into the veins some hours before the necropsy, the lungs do not collapse, and the gross pathologic picture is much more true than when a collapsed lung is studied. In lungs studied in this way the triangular area of hepatization corresponds exactly to the Roentgen-ray shadow. The lobes affected by bronchopneumonia are never completely solid, but show lobular patches separated by more or less aerated lung tissue.

Mason[38] in a preliminary report demonstrated the character of the shadow in lobar pneumonia. He found that the shadow usually developed with the physical signs and cleared up at the same time with them. In all the early cases when the shadow occupied the periphery there was only dulness but when the shadow extended to the bronchi at the hilus, bronchial breathing appeared. Bronchopneumonia gave only a diffuse mottling; no sharp shadows.

Hartshorn[39] reports nine cases of pulmonary conditions, with illustrations showing how the Roentgen ray was useful in clearing up obscure diagnoses. These include pneumonia, tuberculosis, pleural effusions, lung abscesses, and pyopneumothorax. He calls special attention to the value of the Roentgen ray in early diagnosis of pneumonia, in which the shadows often show before the physical signs appear. The Roentgen ray shows the extent more accurately than does the physical examination.

Mouriquand[40] called attention to the diminution of expansion in the infraclavicular region on the affected side in pneumonia. This often lasts eight to fourteen days, and often helps in retrospective diagnosis. The prognosis of pneumonia is better in the cases in which the physical signs are not recognizable until late in the disease. This holds also for the advent of the triangular Roentgen-ray shadow. The frequency of complications he has found to be 7 per cent. of right upper lobe cases, 28 to 30 per cent. of lower lobe cases, while the left upper lobe cases, which are least common, have 60 per cent. complications.

Hutinel[41] holds that it is not enough to diagnose pulmonary inflammation, but that we should also differentiate between lobar and bronchopneumonia. In nurslings bronchopneumonia is the usual form,

38. Mason: Arch. Pediat., 1914, xxxi, 786.
39. Hartshorn, W. M.: Am. Jour. Dis. Child., 1915, ix, 405.
40. Mouriquand, G.: Pathol. infant., 1912, ix, 71.
41. Hutinel, V.: Rev. gén. de clin. et de therap., 1915, xxix, 49.

but it may be pseudolobar in type and resemble lobar in its symptoms, signs, evolution and sharp defervescence. After three years it is rare except as a result of the acute infectious diseases or with a tracheobronchial adenitis. It has a longer course, a more irregular temperature (which yields more easily to hydrotherapy, etc.), and greater dyspnea than lobar pneumonia. The Roentgen ray gives no shadow with bronchopneumonia, while lobar pneumonia casts a definite sharply outlined shadow.

The "abortive" cases which have all the initial symptoms but terminate in a few days with a sudden crisis, may possibly represent a pneumonia which goes only to the stage of congestion, with cure before hepatization. Hutinel has often observed tuberculosis following such an abortive attack. The prognosis of lobar pneumonia is nearly always excellent. It is rarely fatal except when complicated, and since the complication makes the prognosis so much worse, careful watch must be kept for otitis, meningitis, pericarditis, peritonitis, arthritis and nephritis.

Fleischner[42] thinks that the most common reasons for faulty diagnoses in lobar pneumonia are due to its frequent atypical nature, the absence of early signs, failure to make repeated examinations, and the difficulty of eliciting and interpreting signs in childhood. The most common signs are diminution of the breath sounds, increased crying voice, and slight dulness. He emphasizes the importance of light percussion, and the use of the Roentgen ray. He cites cases simulating meningitis, appendicitis, typhoid and empyema.

Pisek[43] in a preliminary report on 1,000 cases of pneumonia gives a mortality of 43.3 per cent. This is very high, but these were all hospital cases which are of the worst type. In the first two years of life bronchopneumonia predominated and was rare after the third year. Lobar pneumonia was much the more common after the third year, except in terminal or secondary infections, and is much more frequent than is usually supposed. Its cause was nearly always the pneumococcus. In the bronchopneumonias many different organisms were found.

Freeman[44] believes that pneumonia is rarely a lobar consolidation, and that bronchopneumonia is much the most common form in children, on account of the larger proportion of bronchi and the accompanying connective tissue. He describes the sequence as first a bronchitis, then peribronchial exudate, forming bronchopneumonic areas

42. Fleischner: Arch. Pediat., 1915, xxxii, 165.
43. Pisek, G.: Arch. Pediat., 1915, xxxii, 389.
44. Freeman, R. A.: Am. Med., 1913, xix, 433.

which may fuse to form a lobar pneumonia. He thinks that in the bronchopneumonias of infancy there is often little bronchitis, and very frequently pleurisy. He does not believe in the usual classification for teaching purposes, but thinks that the distinction between lobar and bronchopneumonia need not be made. Pneumonia might better be presented to students as one disease with a sudden onset, with a persistent high or remittent temperature. The prognosis is good in primary cases in a healthy child. In secondary cases it should be guarded. With a persistent high temperature, pneumococcus infections are most common. With remittent temperature he suspects a mixed infection. With persistent high temperature a crisis may be expected; with remissions a longer course and lysis. The physical examination may or may not confirm diagnosis. Localized crepitant râles are the most common sign.

Thurlings[45] has summarized the whole matter of the existence of lobar pneumonia in infancy. Pathologists all consider that bronchopneumonia is much more frequent in infants and young children. Clinicians, however, agree that lobar pneumonia is not at all uncommon. This difference of opinion is explainable by the mortality statistics. In lobar pneumonia the death rate is from 1.5 to 5 per cent. and it is rarely fatal except in complicated cases. Bronchopneumonia is a very fatal disease and so comes to necropsy much more often in childhood than does lobar pneumonia; and it is on this occurrence on the necropsy table that pathologists base their statistics.

[This view certainly agrees with the usual experience in dispensary and private practice. It is very unusual to have a case come to a dispensary with bronchopneumonia, while lobar pneumonia is brought in every few days. The hospital figures do not represent the incidence of the two diseases because they deal with the worst feeding cases, marasmus, infectious diseases — all conditions which predispose to bronchpneumonia. That lobar pneumonia is not uncommon even in the hospitals, is shown by some recent figures from the Babies' Hospital:[46]

	Cured	Died	Mortality, Pct.
Lobar pneumonia on admission.....	52	1	1.9
Bronchopneumonia on admission.....	30	33	52.5
Bronchopneumonia acquired in hospital	3	13	81.0

In teaching students it is a regular experience, in showing a case of lobar pneumonia, to have it called bronchopneumonia, merely because it occurs in a child. It seems as if the recent evidence should do much to change the teaching of this subject.]

Dilatation of the nostrils in respiration in pneumonia is usually an active muscular phenomenon, according to Carducci.[47] This dilatation

45. Thurlings, M.: Dissertation, Freiburg, 1912.
46. Personal communication from resident physician.
47. Carducci, L.: Ref. in Arch. f. Kinderh., 1915, lxiv, 449.

occurs during inspiration. Passive motion of the nostrils, i. e., collapse during inspiration and dilatation during expiration, is a different matter and is seen only in fatal cases. It is not present in all fatal cases but when it is observed the prognosis is hopeless.

Hess[48] has studied the leukocyte counts in pneumonia. He believes that the primary pneumonias of infancy are lobar and that bronchopneumonia occurs only as a secondary infection or in infants of very low vitality. In spite of the low leukocyte counts in healthy infants, the reaction to pneumonia is very much the same as in adults. The leukocyte count depends on two factors—the reaction of the organism and the severity of the infection. Thus a high count indicates a severe infection and a strong resistance, a moderate count shows a slight infection with a good resistance or a severe infection with a poor resistance, and a low count shows a very slight infection or an overpowering one. A prognosis is impossible from the leukocyte count alone, but when taken in connection with the clinical symptoms it becomes of the greatest significance. The usual blood picture is a very high neutrophil count, and a moderate lymphocyte increase before the crisis. A deviation from this picture points to tuberculosis, typhoid, influenza or rickets. Very high counts should lead to a suspicion of empyema. The neutrophils rapidly disappear after the crisis, but the pseudocrises have no effect on the count. A slow fall with continued fever usually means delayed resolution or complications, but this is the regular behavior in secondary pneumonias. A sudden increase often points to the advent of a complication. The eosinophils disappear during the height of the disease and reappear just before or after the crisis. They have no value in prognosis, but their disappearance following a return makes a complication seem likely. In the secondary pneumonias the count varies and depends largely on the complications. Not infrequently the lymphocytes are high.

Baginsky[49] discusses the treatment of acute lobar pneumonia as a therapeutic paradigm. He cites the histories of several cases of typical pneumonia with crisis and recovery, in which the only treatment was rest, fluid diet and an ice bag to the region of the chest involved. He shows that interfering with the course of the disease is not necessary nor advisable, and greatly deplores the use of digitalis, aspirin, ipecac, apomorphin, camphor, etc., in such cases. Every unnecessary drug is harmful to a child, and also lacks the suggestive influence which comes into the question in adults. In childhood Nature battles more successfully alone. The doctor should watch and help, but only with

48. Hess: Am. Jour. Dis. Child., 1914, vii, 17.
49. Baginsky, A.: Arch. f. Kinderh., 1915, lxiv, 190.

nursing, food, fresh air and cleanliness. The unweakened strength of the child's heart and his ability to manufacture the substances which produce immunity are ample to bring about recovery in the majority of cases. One case is cited which was treated by a younger associate, in which all sorts of needless remedies and therapeutic measures were used, and makes an excellent object lesson of what should not be done. The young physician should remember that the strength wasted in crying and struggling against therapeutic measures might better be saved to combat disease.

Freeman[50] injected six patients with antipneumococcus serum and nine patients with antipneumococcus and antistreptococcus serum. There were four deaths of the first six, and six deaths of the last nine. He concluded that the course of the disease was favorably affected in some cases but that the serum gave no result in most cases. In most of the cases there was a reaction and some improvement. The leukocytes were usually lowered and the polymorphonuclear cells were diminished in number. In the favorable cases there was little spreading of the disease and rapid resolution. The injected cases that recovered had a much shorter course than the controls, and the mortality was slightly less than the control cases. It seemed a safe method of treatment.

[In view of the recent advance of knowledge in regard to the different groups of pneumococci it is not strange that the serums have given such variable and unsatisfactory results. Freeman does not divide the cases into lobar and bronchopneumonias, and this may explain why the results were not better, for it is not to be expected that an antipneumococcus serum would influence a streptococcus bronchopneumonia.]

Delcourt[51] has treated twenty-four lobar pneumonias, and forty-two bronchopneumonias in private practice with continuous inhalations of oxygen. Only one patient with bronchopneumonia died, and the lobar cases all defervesced earlier than usual. It is necessary to be generous with its use, practically letting the child live in an atmosphere of oxygen. The expense is the greatest objection, since four to five thousand liters can be used per day.

SECONDARY AND BRONCHOPNEUMONIA

Hess-Thaysen[52] found that pneumonia of the first few days of life is much more common than formerly supposed. In thirty-two necropsies of children dying in the first three days, pneumonia was found in 14, or 42 per cent. He differentiates possible infections as,

50. Freeman: Am. Jour. Dis. Child., 1912, iv, 383.
51. Delcourt, A.: Path. Infant., 1913, x, 64.
52. Hess-Thaysen: Jahrb. f. Kinderh., 1915, cxxix, 140.

1. Placental infections—luetic infections, tuberculous or rarely pneumococcus, streptococcus or staphylococcus infections.

2. Aspiration pneumonias, either from aspiration of fetid uterine contents or the secretions of an infected birth canal. This corresponds to the old "septic pneumonia." But many cases also occur from aspiration of the secretions of a normal birth canal, or from aspiration of milk or even of mouth secretions.

3. Aerogenous pneumonias may occur, but must be very rare in the early days of life.

4. Metastatic pneumonia from a primary enteritis or navel infection.

The aspiration pneumonias are certainly the most common, especially the form which comes from the aspiration of bacteria containing secretion of the birth canal of a healthy woman. The bacteria found were streptococcus and colon bacillus as a rule. The fact that these pneumonias are found so early points to the fact that they must have begun during or shortly after birth. The clinical diagnosis is impossible, and even at autopsy the inflamed areas are often confused with atelectasis. Microscopic section shows pneumonia in many supposedly atelectatic areas.

Bonnaire and Durante[53] describe the inflammations of the lung due to infections of umbilical origin. They found miliary infarcts, zones of congestion, areas of hepatization, rarely abscesses, the bronchi usually normal. The symptoms are a few scattered râles, slight dulness, slight diminution in breath sounds; cough is rare, but there may be dyspnea or cyanosis. The diagnosis is difficult. An elevation of temperature or its recrudescence after a drop should lead one to investigate the lungs in a new-born, even when there is no manifest infection of the umbilicus. In 309 autopsies with umbilical infection sixty-two cases showed predominating pulmonary lesions. Only forty-one of these were recognized clinically. The umbilicus may appear normal or only very slightly infected.

Bassin[54] reports nineteen cases of pulmonary complications following operations on tonsils and adenoids, the infecton taking place from inhalation of a fragment of tonsil or adenoid or blood clot. This may cause pneumonia, bronchopneumonia, abscess or gangrene of the lung. Great care should be taken to prevent inhalation accidents.

Riesman[55] describes seven cases of bronchopneumonia of a lobar form. The signs were all unilateral—dulness, bronchovesicular breath

53. Bonnaire and Durante: Presse méd., 1913, xxi, 553.
54. Bassin, A.: Thèse de Paris, 1913, No. 181.
55. Riesman, D.: Am. Jour. Med. Sc., 1913, cxlvi, 313.

sounds, and crackling râles. Temperature ran to 101 F. There was slight cough. The duration was several weeks up to four months. The prognosis was good, all recovering completely. These cases were thought to be confluent lobular pneumonia and are perhaps one cause of obscure fevers which is overlooked through careless examination. The protracted course and the fever lead to the suspicion of typhoid or tuberculosis. His treatment was counterirritation, the administration of creosote, a sedative cough mixture, careful feeding, rest and fresh air.

Suner[56] describes cases of bronchopneumonia with which are associated spasmodic symptoms which resemble laryngeal stenosis. The larynx is concerned in the symptoms even though there is no membrane, for the symptoms are relieved by intubation. In his cases streptococcus, pneumococcus and Friedlander's bacillus were the infecting organisms. In one case there was diphtheria of the bronchi but not of the larynx. They mostly occurred following measles or diphtheria. The symptoms were divided into three periods.

The first was a period of initial catarrh. There was a simple coryza, tracheitis, or bronchitis with fever of varying degree. This lasted usually two to nine days, but occasionally only a few hours. The second period was that of laryngeal stenosis. There was gradually increasing hoarseness, the voice became weaker and was gradually lost. Stridor supervenes with increasing dyspnea and cyanosis. The third period was that of asphyxiation. Death occurred if not relieved by intubation or tracheotomy.

This condition should always be considered in a bronchopneumonia, whenever there are symptoms of tracheitis, as it may rapidly go on to asphyxiation. The diagnosis is made by the initial catarrh in the presence of bronchopneumonia, the absence of membrane in the nose, throat or larynx, and the absence of Klebs-Loeffler bacilli from the culture. The prognosis is bad, especially in the rapid cases. Antitoxin should always be given as there is no time to wait for result of cultures. Intubation relieves the symptoms temporarily.

Comby[57] reviews the work of Suner and others and recommends the same treatment, but has found that the intubation allays symptoms only temporarily, because to the very serious inherent dangers of bronchopneumonia are added those of laryngeal stenosis.

Czerny[58] describes paravertebral pneumonias which occur in nutritional disturbances, in rickets and after measles. There are three theories as to the causes of this condition:

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56. Suner: Jahrb. f. Kinderh., 1914, lxxx, 579.
57. Comby, J.: Arch. de méd. des enf., 1914, xvii, 452.
58. Czerny, A.: Jahrb. f. Kinderh., 1913, lxxix, 727.

1. Diminished immunity through nutritional disturbances;
2. Infection through a damaged intestinal wall;
3. Hypostatic congestion predisposing to pneumonia.

Disturbances of nutrition cause abdominal distention and pressure on the diaphragm. This gives lessened diaphragmatic breathing, which causes hypostatic congestion. In pathologic lessening of the intra-abdominal pressure the diaphragm becomes lowered, which also induces disturbed circulation in the lung. Through improved technic of feeding and prevention of inanition, the abnormal intra-abdominal pressure can be prevented and this is the best prophylaxis against paravertebral pneumonia.

Engel[59] found that paravertebral pneumonia occurs most commonly at the back part of the right upper lobe, and at the apex of the right lower lobe. In the left lung it is localized in the middle of the lower lobe, the upper lobe being usually free.

Arneth[60] reports excellent results from the use of hydrotherapy in the treatment of bronchitis and bronchopneumonia. His earlier practice was to use cool packs, with water at room temperature. These were given for a temperature over 40 C. for one-half hour; temperature over 39 C. for one hour; over 38 C. up to one hour and a half; and 38 C. to normal, two to four hours, three times a day. In cases with fever, packs were applied every three hours. In addition to this he laid stress on the necessity for a systematic change of position. The child should be turned on his side and also into the prone position at regular intervals. The prone position is important since the posterior part of the lungs is most affected by bronchitis, especially in helpless infants. It no doubt aids in preventing or removing atelectasis and stasis of the secretions. Strong children who have good muscular power and who react well stand cool packs well. Weaker children cannot stand the load of heat and their power of reacting is lost. Weaker children respond much better to warm baths and by his success with these children Arneth was led to use the warm baths with robust children as well, with surprisingly good results.

Mustard baths are not used at all, as they require much care and are not well borne by the sensitive skin of the child for long. He has practically abandoned medication except turpentine or oxygen inhalations, which are sometimes useful, but should not be used to the exclusion of hydrotherapy.

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59. Engel: Verhandl. der Gesellsch. d. deutsch. Naturforsch. u. Aertze, 1913, p. 10.
60. Arneth: Deutsch. med. Wchnschr., 1913, xxxix, 1868.

His technic for the use of hot baths is as follows:

> Water is used at a temperature of 41 C. (106 F.) and the temperature is kept up by adding hot water from time to time. With a fever up to 39 C. the duration of the bath is ten minutes; over 39 C., the duration is fifteen minutes. The child's temperature is lowered as a rule, but occasionally it is slightly higher immediately after the bath and drops after half an hour to an hour. The skin becomes red in the bath, in some children more than in others. There is rarely sweating, but the relaxed peripheral vessels allow the skin to radiate heat better than when the surface is cold. There seems to be less tendency for the temperature to reach its former height after warm baths than after cold ones. A favorable influence on the lungs and general condition is noticeable. The children do not object to the warm baths as they do to cold. They become less restless or less apathetic, look fresher and go to sleep usually after the bath. There may be slight temporary dyspnea at first in the tub, and after the bath the respiration remains deeper and slightly accelerated. At the end of the bath in stronger children one or two quick splashes with cool water are given to the chest and neck. The child is then dried and put to bed in warm clothing. The weight curves show good effects, children losing little or nothing in these tedious illnesses, probably because they lose less calories in the hot water than when treated with cold. The baths are kept up in convalescence once or twice a day, as it has been found that they prevent relapses. In acute coryza the hot baths are given but without the cold splash and the child is put into a hot pack after leaving the bath, to induce sweating.

Arneth believes that hot baths are a specific therapy for bronchitis and bronchopneumonia through the diminution of fever, the stimulation of expectoration, the prevention of atelectasis, and the tonic effect on the general condition. Since the use of the hot bath he has seldom lost a case, which is significant in these severe illnesses.

Stepp[61] also advocates the use of hot baths in the case of bronchitis and pneumonia in children. He has observed collapse from cold compresses and thinks that a jacket is inappropriate, merely oppressing the child by its weight on the chest. He also uses cold water on the chest, back and arms every two to three hours, except in weak children. He advocates prophylactic washing of the chest and back in the morning with cold water to prevent taking cold.

FIBROSIS AND BRONCHIECTASIS

Fletcher[52] considers that there are three factors in the forming of bronchiectasis; increase in intrabronchial pressure as in pertussis, the softening of the walls of the bronchi, as in bronchopneumonia, and traction on the bronchi as in fibrosis. The child's bronchus is thinner and softer than in the adult and hence is more susceptible to the factors that cause dilatation. Bronchiectasis is an acute condition in bronchopneumonia. It may be recognized by the persistence of coarse râles at the bases after the acute disease has subsided. It is more chronic in

61. Stepp: Ref. Ztschr. f. Kinderh., 1914, vii, 536.
62. Fletcher, H. M.: Clin. Jour., 1913, xlii, 363.

pertussis, and is frequently found, giving coarse râles and changes in the breath sounds over a localized area after the pertussis has ceased. The chief symptom is expectoration, and bronchiectasis should always be suspected when a child coughs up sputum. The râles often disappear entirely after coughing.

Pulmonary fibrosis may be localized or diffuse; the latter is the more common form in children. It may be due to congenital atelectasis, collapse of the lung from pleural effusion, obstruction of the bronchus or severe general bronchitis in young infants. Bronchopneumonia is the main cause, the inflamed areas becoming fibrotic. The prognosis is better in simple fibrosis than in fibrosis with bronchiectasis. There is usually progressive anemia from chronic absorption, lardaceous disease, tuberculosis or occasionally a cerebral abscess. A certain number of patients with bronchiectasis get entirely well. The treatment is hygienic and climatic. Emptying of the bronchiectasis by holding the child in an inverted position and making him cough prevents the absorption of the retained pus and does much to promote a cure. This should be done several times a day. Creosote inhalation seemed to have some value. Surgical drainage of these cavities is rarely successful, since they are nearly always multiple. Resection of a rib to allow collapse of the lung offers a better chance of success.

Miller[63] believes that measles, whooping cough and rickets are the most important causes of pulmonary fibrosis in children. Measles causes a characteristic form of fibrosis, a diffuse peribronchitis with a general widening of the smaller bronchioles. This picture is so characteristic that a skilled pathologist can make a diagnosis of measles as the primary infection. Pertussis distends the alveoli and bronchi causing emphysema and later bronchopneumonia which may become a fibrosis. Rickets predisposes to bronchitis and the softened ribs and weak muscles aid in causing slow recovery and permanent fibrosis. Under treatment, he emphasizes the importance of exercises, especially of the arms, and of making the child cough in an inverted position to empty the cavities. An occasional emetic is also useful in children who are too young to cough. The prognosis is bad; the cases become chronic lung cases, acquire tuberculosis or die of acute respiratory disease. A few grow up and are fairly well.

Hutinel[64] believes that bronchiectasis is not very infrequent in children. Bronchopneumonia damages and infiltrates the mucosa of the bronchi and sclerosis and bronchiectasis follow. It is especially common after pertussis, measles, influenza, chronic bronchitis, adenoids

63. Miller, C.: Practitioner, 1912, xlvi, 619.
64. Hutinel, V.: La clinique, 1913, viii, 50.

which provoke chronic cough, foreign body in the bronchi, compression of the bronchi by lymph nodes or a rupture of the softened lymph node into a bronchus, pleural adhesion, pulmonary sclerosis, tuberculosis, or syphilis. Bronchiectasis occurs in three forms: 1. Large, irregular cavities (usually due to sclerosis or pleural adhesions); 2. Fusiform or elongated cavities (following bronchopneumonia); 3. Cylindrical cavities. Physical signs are dulness, and diminished breathing over a localized area. If the patient takes a slight cold, many râles appear. The Roentgen ray shows an area of increased density surrounding a cavity. Tuberculosis is rarely a cause of bronchiectasis in children, since in them it is more often a caseating process rather than a sclerosing one (except in the case of mediastinal adenopathy). Syphilis, however, with its sclerosing tendency favors the production of cavities, and is often associated with a secondary tuberculosis.

The prognosis is not always bad, as small dilatations in infancy become relatively smaller as the growth of the bronchi takes place and may eventually become insignificant. These patients should have fresh air and live in the country on account of the danger of tuberculosis, and great care should be taken to prevent colds and secondary infections. Respiratory gymnastics and baths are useful.

Milhit[65] calls attention to the fact that bronchiectasis is not rare in children and may give no signs or symptoms until the advent of some acute catarrhal condition. The cavities are cleaner and less suppurating than in adults and tend to disappear with the growth of the lung. Among the causes, but by no means the only one, is hereditary syphilis. He cites three cases, verified by Wassermann, rapidly relieved by antisyphilitic treatment, all signs completely disappearing. The diagnosis is difficult; all were thought to be tuberculosis at first. Diagnosis was made by the absence of tubercle bacilli and the negative von Pirquet, positive Wassermann, and by the Roentgen ray.

Bataille[66] reports twenty-three cases of obliterating adhesions of the pleural cavity. They are most frequently found in the second year, and are the result of a preceding acute or chronic disease of the lung or pleura. The thick sclerotic tissue firmly unites the lung and chest wall. This results in two types of cases, those with deformity of the chest and vertebral column, and those with respiratory and circulatory disturbances due to the involvement of the lung and mediastinum. The diagnosis is difficult as the symptoms simulate all the chronic diseases of the lung and pleura, and can only be made by careful study of the history, signs, and by the Roentgen ray. The prognosis is doubtful and

65. Milhit: Arch. de méd. des enf., 1914, xvii, 105.
66. Bataille: Thèse de Paris, 1913, No. 367.

depends on the amount of mediastinal and lung involvement. The treatment is general hygienic measures, respiratory gymnastics, rest, and heliotherapy.

Lederer[67] believes that the beginning of chronic inflammations of the lungs in later childhood often goes back to infancy, and that many of these are not tuberculous. Their origin is in bronchitis or bronchopneumonia and often follows infectious diseases. The chronic process may end in an acute pneumonia or gradually change into an insignificant condition lasting for weeks or years. There may be complete recovery even after a long time, leaving only a small amount of fibrosis or mediastinal adenopathy. The prognosis is bad when there is extensive induration. The differentiation from tuberculosis can only be made by the course and by the Roentgen ray.

Sutherland and Jubb[68] believe that phthisis is rarer in children than is commonly thought, and that many cases so diagnosed are really chronic pneumococcus infections following pneumonia or bronchopneumonia, which leave a chronic bronchitis, bronchiectasis or fibrosis. They base this view largely on the failure to find tubercle bacilli in the sputum in children (only 9 per cent. positive cases), and on the positive finding of pneumococci in the sputum of many of these patients. They believe that cough with sputum, emaciation, following measles or pertussis should not be enough to make a diagnosis of tuberculosis when the sputum is negative. Even if the skin reaction is positive, it may occur from some other cause.

[The conclusion of these writers is based entirely on theoretical grounds; they give no pathologic evidence; nothing is said of the importance of fever, the character of the physical signs or the use of the Roentgen ray. The finding of the tubercle bacilli in the sputum of children is perhaps the least frequent diagnostic aid in tuberculosis in children.]

Pielsticker and Vogt[69] report ten cases of *artificial pneumothorax* in children. There were six cases of tuberculosis and four of bronchitis. The injection of nitrogen was well borne by most children. Their results were excellent, except in cases in which there were dense pleural adhesions which prevented the collapse of the lung, and this was true especially in the bronchiectasis. The tuberculous cases all did well.

Huber and Berkowitz[70] report a case of primary *actinomycosis* of the lung in a boy of 10. There were signs of consolidation of the upper right lobe, fetid breath and sputum. A diagnosis of gangrene

67. Lederer, R.: Jahrb. f. Kinderh., 1913, lxxviii, 68.
68. Sutherland and Jubb: Brit. Med. Jour., May 31, 1913.
69. Pielsticker and Vogt: Monatschr. f. Kinderh., 1912, xi.
70. Huber and Berkowitz: Am. Jour. Dis. Child., 1914, viii, 113.

was made. Pus was obtained on aspiration and the lung was drained. The child died. The diagnosis was made only at necropsy and the condition was found to be due to the ray fungus.

Mauthner[71] reports a case of chronic bronchitis in a girl of 5. The sputum was greenish and contained a peculiar fungus hitherto undescribed. It was thought that the infection might have come from a pet turtle dove with which the child had been playing.

PLEURAL EFFUSIONS

Delhaye and Lawaese-Delhaye[72] have studied the cell count in pleural effusions. They found that the count does not definitely show the bacterial origin, but depends also on the intensity of the onset, the severity of the infection, and the time of puncture. Pyogenic organisms usually give a neutrophil increase, occasionally a lymphocytosis, and sometimes an eosinophilia. Tuberculosis tends toward a lymphocytosis, but occasionally the polymorphonuclears predominate. The nuclear formula of Arneth has not been higher in coccus infections than in tuberculosis, in their experience.

Weihe[73] has found that *interlobular pleural effusion* is not rare, having observed eight cases in two years. Only one of these was tuberculous, the rest metapneumonic. They occurred between the ages of 5 months and 3½ years. The effusion was small in amount in all cases; 3 c.c. was the largest amount obtained by puncture. The fluid was slightly turbid, due to the presence of leukocytes, but was sterile on culture mediums and animal injection. The diagnosis was made by the Roentgen ray which showed a narrow shadow, usually between the right upper and middle lobes. There was fever which suggested an empyema, but in no case was pus found. All of the patients recovered.

Mouriquand[74] calls attention to the importance of dulness at the base of the axilla in pneumonia. The percussion of pneumonia at the base, no matter how extensive, rarely gives dulness in the axilla or anterior part of the chest. Only the massive pneumonias give this dulness, and these are rare in infancy. A very small amount of fluid, however, gives axillary dulness. In all of thirty cases with this dulness, fluid was found on exploration. In cases in which resonance persisted at the base of the axilla, even when the auscultatory signs pointed to fluid, none was found on puncture.

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71. Mauthner, V.: Arch. f. Kinderh., 1914, lxxx, 341.
72. Delhaye and Lawaese-Delhaye: Bull. Soc. méd. d'Anvers., 1913, lxxiv, 185.
73. Weihe, F.: Ztschr. f. Kinderh., 1915, xiii, 119.
74. Mouriquand, G.: Pathol. inf., 1913, iv, 68.

EMPYEMA

Holt[75] reported 154 cases of empyema in children under 3 years of age. The mortality was 56 per cent., which seems high, but it must be remembered that these were all very young patients. The importance of age as a factor is shown by the mortality for each year.

First year, mortality.....73 per cent.
Second year, mortality.....58 per cent.
Third year, mortality.....16 per cent.

The infecting organism influences the mortality greatly.

Streptococcus infections, mortality.....79 per cent.
Mixed infections, mortality.....62 per cent.
Pneumococcus infections, mortality.....56 per cent.
Staphylococcus infections, mortality.....50 per cent.

The leukocyte count was found to be high, as a rule, but varied from 6,000 to 81,000. There was no relation between the blood count and the type of infection, and it had no value in prognosis. The cause of death was usually sepsis and exhaustion, or some secondary infection, as pericarditis, meningitis, secondary pneumonia. Most of the cases were treated by simple incision, rib resection, or siphon drainage. Aspiration, and aspiration followed by the injection of various substances, were used in a smaller number, but the latter plan seemed to have no advantages.

Siphon drainage is the method of choice in the first year. The technic perfected by Kenyon was used. An exploratory aspiration is first performed. An incision is made with a small scalpel, large enough to permit the insertion of a rubber tube (size 27 French) around which is a collar to prevent slipping into the chest. The tube is strapped tightly to the chest wall with adhesive plaster, so as to make a tight joint, and is connected by means of a long rubber tube to a bottle under the bed. This bottle has a glass tube extending nearly to the bottom, and is kept half full of sterile salt solution, so that air can not enter the chest. The discharge is easily measured in this way, and varies from one to eight ounces a day. If the tube blocks, it may be cleared by "milking," or by raising the bottle and allowing the previously warmed salt solution to flow into the chest. This method has the advantage of avoiding the daily dressing with the struggling and fear of pain which it causes. It is a small operation and can be done under ethyl chlorid, local anesthesia, or even with no anesthetic. It favors expansion of the lung by preventing the entrance of air into the pleural cavity. Atmospheric pressure seems to prevent lung expansion in young infants. There is also less danger of secondary infection

75. Holt, L. Emmett: Am. Med., 1913, viii, 381.

of the pleura with a sealed opening. If leakage of air takes place around the tube from ulceration, this plan may have to be abandoned and simple drainage used.

Next to siphon drainage, simple incision is best in infants. Rib resection is not necessary except in chronic cases or as a secondary operation where simple incision does not give good drainage. Aspiration occasionally cures a small empyema but is not to be relied on, and there is no advantage in injecting bactericidal substances into the pleural cavity.

Of the cases under 1 year there were:

Simple incision	16 cases, 3 recoveries
Rib resection	9 cases, 2 recoveries
Siphon drainage	20 cases, 8 recoveries

Savoriaud[76] gives a general summary of the symptoms and course of empyema and lays stress on the fact that the pneumonic are relatively more benign than the streptococcic infections. He gives the following indications for treatment: In tuberculous cases, aspiration only, as in a cold abscess, for incision means mixed infection and a persistent fistula. In mixed infections he recommends incision. In pneumonic effusions which have opened spontaneously into a bronchus, the treatment should be expectant. In pneumococcic empyema he recommends puncture if the case is mild, which may be repeated once, but if a third puncture is necessary, or if the patient does badly after the first puncture, it is better to incise. Staphylococcus or streptococcus infections or any fetid or gangrenous effusions must be incised at once. Incision is indicated whenever there are signs of septicemia. He describes only the technic of rib resection and evidently considers it the only form of operation. He does not give any figures to show his results.

In Dunlop's[77] experience empyema occurred in about 10 per cent. of the cases of pneumonia. He had a mortality of 19 per cent.; under two, 36 per cent.; over two, 13 per cent. Pneumococcus infections over two all recovered. He found the following organisms: pneumococcus, 53 per cent.; streptococcus, 16 per cent.; mixed infection, 14 per cent.; staphylococcus, 3 per cent.; tubercle bacillus, 3 per cent.

He lays stress on the point that latency of empyema usually means carelessness of the physician, and insists on the necessity for careful examination after pneumonia. The diagnosis must be made mainly by the percussion notes. The breath sounds are less reliable and the vocal fremitus is worthless in children as a diagnostic sign. He recommends aspiration in the very young, and also in very large effusions

76. Savoriaud: Jour. de méd. de Paris, 1913, xxxiii, 498.
77. Dunlop, H. G. M.: Edinburgh Med. Jour., 1914, xiii, 4.

the day before the operation so that a large amount of fluid need not be evacuated at the operation. Simple incision is good in very young children, as there is little shock and usually good drainage. His results:

Aspiration	8 cases, 1 death
Resection	61 cases, 14 deaths
Incision	29 cases, 5 deaths

Dowd[78] collected 285 cases of empyema with a mortality of 25 per cent. He divides the surgical treatment into diagnostic aspiration, methods of pus removal, treatment of chest after thoracotomy, treatment of complications. Aspiration for diagnosis requires a large needle and a good syringe. This seems simple, but good syringes are not by any means easily obtainable. Aspiration is not free from danger, as 25 deaths have been reported from this account alone. If one lung is incapacitated, aspiration of the opposite side may cause a pneumothorax and death. The pus removal is not a difficult operation. Ether should not be used as it is not well borne in pneumonia. Under a local anesthetic (novocain 1 per cent.) simple incision or rib resection may be easily done. Dowd prefers resection except in very young children. As soon as the pus is evacuated a coughing attack should be provoked if possible by irritating the pleura. The drainage of the chest is best promoted by a flanged spool, long tubes being inadvisable. A suction apparatus is not necessary. Blowing exercises during convalescence aid greatly in expanding the lung. Roentgenograms should be taken from time to time as there is frequently a secondary pneumonia or empyema of the other side. The danger of pocketing is much exaggerated as pockets very rarely occur. Northrup has never seen pus pocketed in a drained pleural cavity. A good deal of harm may be done in searching for pockets which do not exist, the complication really being pneumonia. A compressed lung which does not expand is the most serious complication next to pneumonia. If forced coughing and blowing exercises do not expand it, an enlarged thoracotomy and decortization of the lung may be done, and often effect a cure. Bronchial fistula may close spontaneously, or may require thoracoplasty and lung decortization.

Buttermilch[79] believes that puncture is better than rib resection in infants. The prognosis does not depend on the treatment entirely, but more on the course of the preceding or accompanying disease. The difference in malignancy depends on the virulence of the bacteria:

Simple puncture	3 cases, 2 deaths
Puncture and then resection	3 cases, 3 deaths
Resection	8 cases, 2 deaths

78. Dowd, C. G.: New York State Jour. Med., 1914, xiv, 342.
79. Buttermilch, V.: Ref. in Ztschr. f. Kinderh., 1913, vii, 102.

Stettin's[80] indications for operation depend on the virulence of the infection and on the strength of the patient. He advises rib resection in a strong child with a thick pus or when fluid reaccumulates after puncture. He uses puncture only in underweight or atrophic children, where there is very thin pus, in double empyema, or in polyserositis. Discussing Stettin's paper, Rietschl points out that so long as there is a pneumonia present, thoracotomy means death. Punctures should be used until the patient has recovered from the pneumonia, and then thoracotomy if necessary.

Hahn[81] recommends siphon drainage in children, especially in double empyema. He reports 80 per cent. cured with an average duration of twenty-five days. If the discharge persists after six weeks, rib resection should be done especially in sacculated empyema.

Werner[82] reports 178 cases of rib resection and drainage with 21 per cent. mortality, most all of which occurred in the first two years. He found that the pneumococcus infections had the best prognosis, mixed infections the worst. In a few cases he resected two ribs but the results were poorer and the healing slower.

Zingher[83] reports a case of bilateral empyema following acute bronchopneumonia in a child of 6½ years. The operation on the left side was done five weeks after the onset; on the right side eight weeks. The child recovered. He believes that the interval between opening both chests in empyema may be made shorter than is usually supposed. It has been done simultaneously with recovery. If only one side is done at a time, the left should be done first.

Nobécourt and Saint Girons[84] report an upper lobe empyema in a 14 month's child. It followed a mild pneumonia. There was fever, and dulness and signs of consolidation with Roentgen-ray shadow down to the nipple. On the fourth puncture pus was obtained which was dark colored, thick and showed pneumococci. Upper lobe empyema is rare in children. Only twenty-five cases in literature and only three in infants have been reported. The prognosis is good as all twenty-five recovered, with only one resection.

SUBCUTANEOUS EMPHYSEMA

Comby[85] gives the following causes of subcutaneous emphysema occurring in the course of acute respiratory disease: Lesions of the nasal fossae, the accessory sinuses, larynx, trachea (intubation or tracheotomy), foreign body or rupture of the bronchi, rupture of

80. Stettin, E. H.: Ref. in Ztschr. f. Kinderh., 1913, vii, 101.
81. Hahn, B.: Deutsch. med. Wchnschr., 1913, xxxix, 1830.
82. Werner: Deutsch. Ztschr. f. Chirurg., 1913, cxxiv, 411.
83. Zingher, A.: Arch. Pediat., 1913, xxx, 684.
84. Nobécourt and Saint Girons: Ref. Ztschr. f. Kinderh., 1914, vii, 215.
85. Comby, J.: Arch. de méd. d. enf., 1914, xvii, 81.

mediastinal lymph node, rupture of the lung tissue or of a subpleural tubercle. Rarely it may be caused by a lesion of the esophagus, stomach or intestine. The air penetrates the mediastinum, reaches the root of the neck and extends to the face and trunk. It is nearly always accompanied by dyspnea, cyanosis and coldness of the extremities, and often by fever. It may gradually disappear in from one to two weeks, or may cause death from asphyxia. The prognosis is bad in most cases, especially when there is tuberculosis or bronchopneumonia. The diagnosis of the emphysema is easy, no other condition giving the soft swelling with crepitation, but the diagnosis of the cause is more difficult and can only be made from the course and evolution of the primary disease. There is no treatment for the emphysema itself, but the dyspnea may need stimulation (digitalis or theobromin), oxygen, external heat to the extremities, antispasmodics, purgation, etc.

Delcourt[86] reports a case of bronchopneumonia of the pseudolobar type following measles, in which subcutaneous emphysema appeared on the neck and thorax. It gradually disappeared in about eight days and the child recovered. This is a rare outcome in emphysema with bronchopneumonia.

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86. Delcourt, A.: Pathol. infant., 1913, x, 64.

CURRENT PEDIATRIC LITERATURE

HYGIENE

Open-Air Schools, Medical Aspects of.—Sherman C. Kingsley.
Jour. Am. Med. Assn., Oct. 30, 1915.

BACTERIOLOGY AND PATHOLOGY

Diphtheria Bacilli, Economical Intracutaneous Method for Testing Virulence of.—A. Zingher and D. Soletsky.
Jour. Infect. Dis., November, 1915.

Hypopituitarism; Report of Two Cases.—L. N. Boston.
New York Med. Jour., Oct. 30, 1915.

Myxedema, with Tetany and Suprarenal Insufficiency; Case of.—J. Eason.
Edinburgh Med. Jour., October, 1915.

Status Thymicus Associated Probably with Inferior Thyroid Lobe.—H. A. Houghton.
New York Med. Jour., Oct. 30, 1915.

Streptococci, Elective Localization of.—Edward C. Rosenow.
Jour. Am. Med. Assn., Nov. 13, 1915.

METABOLISM AND NUTRITION

Albumin Milk and its Substitutes.—H. Glanzmann.
Jahrb. f. Kinderh., October, 1915.

Carbohydrate Metabolism, Studies in: Influence of Hydrazin on Respiratory Quotient and on Heat Production.—F. P. Underhill and J. R. Murlin.
Jour. Biol. Chem., October, 1915.

Feeding, Infant, Artificial.—J. Epstein.
Med. Rec., New York, Nov. 13, 1915.

Lactation, Deficient, Management of.—N. S. Heaney.
Surg., Gynec. and Obst., November, 1915.

Milk, Antagonism Between Lactic-Acid and Spore-Bearing Organisms in.—W. S. Kiester.
Bull. Johns Hopkins Hosp., October, 1915.

Milk in Diet of Infants.—D. L. Smith.
Jour. South Carolina Med. Assn., October, 1915.

Milk, Pasteurization of.—J. Race.
Pub. Health Jour., October, 1915.

Obesity, Pathologic, in Two Infants.—L. Ferrannini.
Riforma med., Oct. 2, 1915.

Tetany, Etiology of — Metabolic and Clinical Studies.—A. Brown and A. Fletcher.
Am. Jour. Dis. Child., November, 1915.

DISEASES OF THE NEW-BORN

Developmentally Deficient Children Twice Born to Same Couple.—W. C. Gayler.
Am. Jour. Obst., October, 1915.

Hemorrhage of the New-Born, A Simple Method of Transfusion in, with Report of a Case.—W. S. Howard.
Jour. Am. Med. Assn., Oct. 16, 1915.

Ophthalmia Neonatorum, Some Aspects of.—George S. Derby.
Jour. Am. Med. Assn., Oct. 16, 1915.

Prenatal Care. Deductions from the Study of Three Thousand Four Hundred and Sixteen Cases.—Mary Lee Edward.
Jour. Am. Med. Assn., Oct. 16, 1915.

ACUTE INFECTIOUS DISEASES

Cerebrospinal Fever, Etiology and Prevention of.—H. Sutherland.
Lancet, London, Oct. 16, 1915.

Diphtheria, and other Pharyngeal and Nasal Conditions, Results of Examinations of Cultures from Suspected Cases of.—G. R. Moffitt.
Pennsylvania Med. Jour., October, 1915.

Diphtheria, Incubation Time in Culture Diagnosis of.—A. Gehrmann.
Am. Jour. Pub. Health, October, 1915.

Diphtheria, Local, Bacillus-Broth-Culture Reaction in Man.—G. Bessau and J. Schwenke.
Monatschr. f. Kinderh., 1915, xiii, No. 9.

Diphtheria, Municipal Control of.—W. G. McKay.
Am. Jour. Pub. Health, October, 1915.

Fevers, Obscure, of Infancy and Childhood.—B. K. Rachford.
Lancet-Clinic, Oct. 2, 1915.

Measles at Naval Training Station, Epidemiology of Outbreak of.—C. E. Riggs.
U. S. Navy Med. Bull., October, 1915.

Measles, Transmission and Administrative Control of.—H. F. Gray.
Jour. Infect. Dis., November, 1915.

Meningitis, Acute, Following Antityphoid Vaccination.—Moussaud and R. J. Weissenbach.
Ann. de gynec. et d'obst., September-October, 1915.

Meningitis, Atypical Acute.—Lacoste and L. Boidin.
Ann. de gynec. et d'obst., September-October, 1915.

Meningitis, Cerebrospinal, Abortive Forms of.—H. Hochhaus.
Deutsch. med. Wchnschr., Sept. 30, 1915.

Meningitis, Cerebrospinal.—A. Kuhlmann.
Journal-Lancet, Oct. 15, 1915.

Meningitis, Early Diagnosis of Cerebrospinal.—E. Weill.
Lyon méd., 1915, cxxiv, No. 4.

Meningitis, Influenzal.—C. V. Boland.
Lancet, London, Sept. 25, 1915.

Meningitis, Influenzal; Report of Case.—F. A. Ely.
Jour. Iowa State Med. Soc., October, 1915.

Meningitis, Serotherapy in Epidemic Cerebrospinal.—M. Kurak.
Med. Klin., Sept. 19, 1915.

Meningitis, Streptothrix Cerebrospinal in Six-Months Infant.—G. Rutelli.
Pediatria, Naples, October, 1915.

Pertussis Vaccine as Prophylaxis; Report of its Use as Such.—S. C. Frankel.
Kentucky Med. Jour., September, 1915.

Poliomyelitis, Epidemiologic Study of.—W. A. Sawyer.
Am. Jour. Trop. Dis. and Prev. Med., September, 1915.

Poliomyelitis, Paralysis of Abdominal Muscles with Acute Anterior.—K. Halbey.
Med. Klin., Sept. 19, 1915.

Poliomyelitis, Roentgenographic Bone Changes in a Case of.—G. B. Hassin.
Jour. Am. Med. Assn., Oct. 23, 1915.

Poliomyelitis, Spinal Fluid in, and its Differentiation from Fluids of other Infections.—H. L. Abramson.
Am. Jour. Dis. Child., November, 1915.

Scarlet Fever, Influence of Summer Vacation on Mortality from Diphtheria and.—L. Bernhard.
Arch. f. Kinderh., 1915, lxiv, Nos. 5-6.

Schick Test.—J. Zuckerman.
New York Med. Jour., Oct. 16, 1915.

Should Public Schools be Closed when Epidemic Develops?—F. G. Boudreau.
Ohio State Med. Jour., September, 1915.

Tetanus in Boy of 4; Recovery under Serotherapy and Magnesium Sulphate.—J. Nordentoft.
Hospitalstidende, Sept. 22, 1915.

Tonsils, Relation of, to Rheumatism.—W. Johnston.
Northwest Med., October, 1915.

Typhoid in Children, Analysis of Three Hundred and Eight Cases of.—K. G. Percy.
Boston Med. and Surg. Jour., Oct. 14, 1915.

Varicella, Prophylactic Vaccination for.—S. Rabinoff.
Arch. Pediat., September, 1915.

Whooping Cough, Paralysis with; Four Cases.—S. T. Sörensen.
Arch. f. Kinderh., 1915, lxiv, Nos. 5-6.

Whooping Cough, the Bacillus of.—I. Jacobsen and A. H. Meyer.
Arch. f. Kinderh., 1915, lxiv, Nos. 5-6.

Whooping Cough, Statistical Study of.—F. S. Crum.
Am. Jour. Pub. Health, October, 1915.

Whooping Cough, Treatment should be Addressed to the Nervous System.—V. Lange.
Berl. klin. Wchnschr., Oct. 11, 1915.

TUBERCULOSIS AND SYPHILIS

Bovine Infection in Child, Relation of, to Clinical Tuberculosis in Adult.—F. M. Pottenger.
South. Med. Jour., November, 1915.

Hip Disease, Quiet.—H. L. Taylor and W. Frieder.
Am. Jour. Orthop. Surg., October, 1915.

Meningitis, Tubercular.—R. M. Politzer.
Jour. South Carolina Med. Assn., October, 1915.

Syphilis, in Feeblemindedness, Inherited.—W. H. McKay.
Illinois Med. Jour., October, 1915.

Syphilis, Primary of Tonsil.—C. M. Smith.
Jour. Cutan. Dis., October, 1915.

Syphilis We See But Do Not Recognize. Importance of Familial Studies.—H. F. Stoll.
Boston Med. and Surg. Jour., Oct. 21, 1915.

Tuberculosis in Childhood, What Constitutes.—J. L. Morse.
Boston Med. and Surg. Jour., Oct. 28, 1915.

Tuberculous Exposure Controlled by Von Pirquet Reaction, Children in Relation to.—J. B. Manning and H. J. Knott.
Am. Jour. Dis. Child., November, 1915.

Tuberculous Infection, Childhood Susceptibility to.—J. H. Peck.
Jour. Iowa State Med. Soc., October, 1915.

Tuberculous Meningitis; Prevalence in Children of Communities Frequented by Tuberculous Health Seekers.—R. E. Thomas.
Arizona Med. Jour., October, 1915.

Tuberculosis, Method of Dealing with, in Public Schools of Toronto.—F. S. Minns.
Canad. Med. Assn. Jour., October, 1915.

Tuberculosis, The Initial Fever of.—H. Koch.
Ztschr. f. Kinderh., July 26, 1915.

Tuberculin Treatment of Children.—H. Koch.
Ztschr. f. Kinderh., July 26, 1915.

GASTRO-INTESTINAL SYSTEM

Diarrhea in Infancy.—C. K. Johnson.
New York Med. Jour., Nov. 6, 1915.

Intestinal Anomalies, Congenital.—M. G. Sturgis.
Surg., Gynec. and Obst., October, 1915.

Intestine, Congenital Malformation of.—L. M. Drennan and H. C. Clark.
Am. Jour. Dis. Child., November, 1915.

Intestines, Invagination of the, in Infants and Small Children.—H. Wollin.
Jahrb. f. Kinderh., October, 1915.

Intestinal Parasites in Children.—G. J. Greil.
Am. Jour. Dis. Child., November, 1915.

Intussusception, Early Diagnosis of.—Irving M. Snow.
Jour. Am. Med. Assn., Oct. 30, 1915.

Pyloroplasty, New Methods of, for Congenital Pyloric Stenosis.—Alfred A. Strauss.
Jour. Am. Med. Assn., Oct. 30, 1915.

RESPIRATORY SYSTEM

Bronchopneumonia, Purulent Otitis Media and Interna and Leptomeningitis; Case of; Postmortem; Microscopic Examination of Ear.—J. Thomson and J. S. Fraser.
Edinburgh Med. Jour., October, 1915.

Pleurisy, Interlobar, in Children and the Roentgen Findings.—F. Weihe.
Ztschr. f. Kinderh., July 26, 1915.

BLOOD AND CIRCULATORY SYSTEM

Blood, Anomalies in the, of the Prematurely Born and Weakly Children.—D. Kunckel.
Ztschr. f. Kinderh., July 26, 1915.

Bradycardia from Heart Block in Boy of 9.—A. d'Espine and Cottin.
Rev. méd. de la Suisse romande, September, 1915.

Heart in Some Acute Infectious Diseases.—W. H. Witt.
Jour. Tennessee State Med. Soc., October, 1915.

Heart, Physical Examination of in Children.—J. E. Talley.
Arch. Pediat., September, 1915.

Hemorrhagic Conditions in Infants, Cases of.—J. Phillips.
Cleveland Med. Jour., August, 1915.

NERVOUS SYSTEM

Chorea, Form of Reflexes in; Technic of Elicitation; Studies in Neurologic Technic No. 4.—W. B. Swift.
Albany Med. Ann., October, 1915.

Convulsions, Spasmophilic, in Infancy; Their Differentiation and Treatment.—C. G. Grulee.
Jour. Michigan Med. Soc., November, 1915.

Delinquent Boys and Girls, Mental Examination of.—T. H. Haines.
Illinois Med. Jour., October, 1915.

Imbecile, High, Psychoses of.—H. J. Berkley.
Am. Jour. Insan., October, 1915.

Massage and Remedial Exercises in Treatment of Children's Paralyses; Their Differentiation in Use.—J. M. Colby.
Boston Med. and Surg. Jour., Nov. 4, 1915.

Mental Deficiency, Available Fields for Research and Prevention in.—M. G. Schlapp.
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Jour. Abnorm. Psychol., October-November, 1915.

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